PATENT APPLICATION

IN THE UNITED STATES PATENT & TRADEMARK OFFICE

In re application of)
)
Applicant: Rosskopf et al.
)
Title: Methods of Reducing Pest By Use)
Of Iodacetic Acid, Bromoacetic)
Acid, 2-Iodoacetamide, or)
2-Bromoacetamide)
)
) Examiner:
Serial No.: 10/828,802)
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PETITION FOR EXTENSION OF TIME

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

Applicants hereby petition for a One month extension of time to respond to the Non-Final Rejection with a mail date of December 28, 2006, thereby extending the period of response to April 28, 2007. Please charge the $110.00 fee for said extension to Deposit Account 50-2134.

Respectfully submitted,

4/19/07
Date

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because rabbit heart muscle shows a marked decrease in total acid-soluble phosphorus and organic phosphorus (95). Plasma levels of other important electrolytes are found to be elevated, particularly just before death occurs. Foss has reported changes in plasma potassium from control levels of 17 mgm./100 cc. to 25 mgm./100 cc. after poisoning has progressed (52). Others report minor increases in potassium and calcium (95). These electrolyte changes are probably nonspecific and reflect the morbidity of cells weakened by an attack at some other point; similar electrolyte alterations occur under many conditions.

IV. THE INTIMATE MECHANISM OF ACTION

A great deal of illumination has been cast upon the manner in which a dose of a fluoroacetate compound brings about the death of an animal. Having been assured that the heart or central nervous system is ultimately disabled by events familiar to pharmacologists, one may properly inquire, "How do these events come about"? Indeed, this is currently the most interesting aspect of the fluoroacetate problem.

There is no longer any reason to believe that fluoroacetate is in any way pharmacologically analogous to iodoacetate, although this was a natural *a priori* assumption. The behavior of poisoned animals, the chemical character unrelated to iodoacetate and the failure of low concentrations to inhibit significantly any system sensitive to iodoacetate seem sufficient reasons to discard this concept entirely. In addition, even though the chemical nature of fluoroacetate suggests no basis for the assumption that fluoride ion might be split off and be the cause of the toxic action of fluoroacetate, the actions of the fluoride ion are entirely different from those of fluoroacetate. One must of necessity conclude that the fluoroacetate ion exerts its action as such and not because of any obvious chemical reactions with it or because of toxic breakdown products.

British workers have succinctly summarized their earlier efforts to identify the systems attacked by fluoroacetate as follows: "No enzyme system has been found which is inhibited to any extent by methyl fluoroacetate" (96). In a most important communication in 1947 describing work carried on in 1944, Bartlett and Barron (12) have reported experiments on the metabolism of animal tissues from which they conclude that, "Fluoroacetate probably acts by inhibiting the formation of "active" acetate (the so-called C_2 compound, which may be an acetyl derivative or an acetate radical)". Because of the importance of this provocative conclusion, it is appropriate to review the observations upon which it is based as well as some experiments which do not justify this conclusion.

A. Tissue Slices, Homogenates and Microorganisms

It was found that 0.001 to 0.2 M fluoroacetate decreases the oxygen uptake of tissues. Tissue slices from rats moribund after an LD_{50} of fluoroacetate were found to oxidize acetate 20 to 30% less vigorously than control slices. The degree of inhibition of acetate oxidation could be increased by adding fluoroacetate to normal tissues *in vitro*; inhibitions as great as 90% occurred in heart slices after 0.005 M fluoroacetate was added. This inhibition might be interpreted as occurring *pari passu* with cellular morbidity except for the fact that the inhibitions of O_2 uptake of guinea pig brain (which does oxidize acetate and does convulse) and of rabbit brain (which does not oxidize acetate and does not ordinarily convulse) were found to be 53% and 11%, respectively.

Experiments were then performed to ascertain the mechanism of acetate blockade and several known routes of pyruvate and acetate metabolism were tested. (1) *Pyruvate→ acetate:* The oxygen consumption of kidney slices in the presence or absence of pyruvate was decreased to about the same extent (60%) by 0.01 M fluoroacetate; yet in the presence of pyruvate, which apparently was oxidized, acetate accumulated. It is apparent that the further oxidation of this acetate had been inhibited. (2) *Acetoacetate ⇌ acetate:* The utilization of acetoacetate by rat kidney slices was completely inhibited by 0.02 M fluoroacetate. Conversely, the formation of acetoacetate from acetate was accelerated by fluoroacetate and this was still further increased as the proportion of oxygen in the gas phase was increased, reaching 233% of normal in pure oxygen. One may infer from this that acetoacetate is converted *in toto* to acetate, the further oxidation of which is inhibited so that acetate accumulates as the end-product of the reactions: glucose → pyruvate → acetate and forces the reaction: acetate → acetoacetate. (3) *Pyruvate → acetate → succinate:* The formation of succinate from pyruvate was 77% inhibited by 0.02 M fluoroacetate. Since none of the individual reactions of the Krebs' tricarboxylic acid cycle was found to be affected by fluoroacetate (see also (84) for a statement of disagreement), this may be interpreted to indicate that pyruvate enters the Krebs' cycle in large part through acetate and not directly. (4) *Other reactions:* Pyruvate and lactate formed by deamination of alanine accumulate in rat kidney slices in the presence of fluoroacetate, a fact that might be readily predicted if it were established that oxidation of pyruvate through acetate is inhibited. Glucose metabolism is directed toward lactate (aerobic glycolysis) to the extent of nearly one half, but the anaerobic utilization of glucose is completely unaffected in kidney slices. Aerobic oxidative synthesis of carbohydrate from pyruvate and acetate is markedly inhibited by fluoroacetate; this fact suggests that, as the entry of acetate into Krebs' cycle is inhibited, this cycle may be a pathway for the synthesis of carbohydrate from pyruvate and acetate. Anaerobic conversion of pyruvate to lactate and acetate was not affected (see also 26). (5) *Acetylation of foreign amines:* The acetylation of sulfanilamide and of p-aminobenzoic acid by rabbit liver slices was increased by 0.02 M fluoroacetate. (This has been confirmed *in vivo* in rats and rabbits (37, 123).) The chemical reaction of acetylation is not affected; formation of acetylcholine from choline in the presence of glucose or pyruvate is not affected by 0.02 M fluoroacetate (see also 85). Therefore, it may be assumed that the increased acetylation of foreign amines is the result of an inhibition of acetate metabolism in consequence of which more acetate becomes available for acetylations.

Having been thus provided with a basis for the concept that fluoroacetate interferes with the oxidation of acetate in various animal tissues, Kalnitsky and Barron (77) studied the details of the phenomenon in baker's yeast and bacteria.

The oxidation of acetate by baker's yeast was 95% inhibited by 0.001 M fluo[ro] acetate (30% by 0.00001 M), only 5% by 0.001 M bromoacetate and not at all by 0.001 M chloro- and iodoacetate; the specificity of the reactions involved is thus demonstrated. Fluorobutyrate and fluorocrotonate (0.001 and 0.003 M) had no inhibitory action at all. The oxygen uptake of yeast suspensions was nearly completely inhibited by fluoroacetate added 15 minutes before acetate and practically unaffected when the two were added together. Appreciable reversal of such fluoroacetate-induced inhibition could be obtained by adding higher concentrations of acetate (0.1 M). Additional evidence for the specificity of the inhibition was obtained when it was found that acetate oxidation by specially washed yeast was completely inhibited by 0.00075 M fluoroacetate while pyruvate oxidation was only 79% inhibited.

By the use of a different approach, it was reasoned that if fluoroacetate is a specific inhibitor of acetate oxidation there should be no immediate inhibition of the O_2 uptake associated with the oxidation of ethanol to acetate through acetaldehyde. This was found to be the case; oxidation of ethanol by baker's yeast in the presence of 0.01 M fluoroacetate progressed exactly as in the absence of fluoroacetate until the accumulation of unoxidized acetate affected the rate of oxidation of ethanol to acetate. Less complete inhibition of acetate oxidation produces less block of ethanol oxidation; Black and Hutchens (15) found that 0.001 M fluoroacetate did not completely prevent the continuation of ethanol oxidation through acetate.

The anaerobic dissimilation of pyruvate to acetate and formate by *Escherichia coli* was unaffected by 0.01 M fluoroacetate although the oxidation of pyruvate to acetate by this organism was definitely inhibited. *Neisseria gonorrheae* does not dissimilate pyruvate but does oxidize it directly to acetate. This reaction is 40% inhibited by 0.01 M fluoroacetate, 26% by 0.02 M acetate and 52% by both together. These experiments add emphasis to the view that inhibition of pyruvate oxidation by fluoroacetate is due to the accumulation of acetate, since even acetate alone is slightly inhibitory.

Quite different results were obtained when another microorganism, *Corynebacterium creatinovorans*, was studied. An *increase* in the endogenous respiration of this organism was produced by both fluoroacetate and fluorobutyrate. It was suggested that this is the result of diversion of cellular metabolism by fluoroacetate toward oxidative pathways in a manner similar to that produced by low concentrations of cyanide and azide. Although acetate oxidation by this organism was completely inhibited by fluoroacetate, fluorobutyrate had no effect at all.

As was mentioned, the greatest inhibition of acetate oxidations (as measured by decreased O_2 uptake) by fluoroacetate occurred when fluoroacetate was added to the yeast some minutes prior to the addition of the acetate substrate. After two hours the inhibition apparently decreased considerably (as measured by increased O_2 uptake), a fact which Kalnitsky and Barron interpreted as the indirect result of the slow accumulation of citrate and its increasing movement into the carboxylic acid cycle. The same characteristic decrease of inhibition was noted

the growth curves of *Tetrahymena geleii* in a glucose but not in an acetate medium (48). A further study of the effect of fluoroacetate upon citrate formation was later made by Kalnitsky (75, 76). Using rabbit kidney cortex homogenate, he found that not only fluoroacetate but barium and magnesium salts as well appear to increase the formation of citrate from oxaloacetate. The effect of barium and magnesium is the result of inhibition of citrate utilization. On the other hand, the slight inhibition of citrate utilization (at fumarate? (84); *v.i.*) produced by high concentrations of fluoroacetate can only account for about 5% of the accumulation of citrate in the presence of fluoroacetate. It was concluded that the increased citrate content might be the result of the inhibition of acetate oxidation reflected through a series of reversible reactions in a manner analogous to the effect of malonate on pyruvate oxidation.

Another approach to the problem of the greater inhibition of fluoroacetate on acetate oxidation by yeast when the fluoroacetate was added before the acetate was made by Black and Hutchens (15). Working in different laboratories, they confirmed the general fact that a more prolonged inhibition of acetate oxidation is obtained by allowing a longer period to elapse between the addition of the inhibitor and the substrate. They suggest that this is similar to the delay noted by Lynen (91) in starved yeast before acetate oxidation becomes vigorous and that it is the result of cellular depletion of certain substances found by Lynen to be essential for acetate oxidation in yeast. Ethanol was found to be particularly efficacious in accelerating the oxidation of acetate by yeast either untreated or pretreated with fluoroacetate.

When calculations of the oxygen consumption of yeast were made using the final rate after equilibrium has been reached, Black and Hutchens found that pyruvate oxidation is more sensitive to fluoroacetate than is acetate oxidation. Because the delay in oxidizing pyruvate is less than for acetate, these workers decided that the earlier conclusion of Kalnitsky and Barron that acetate oxidation is more sensitive than pyruvate was the result of an error in technic. Hutchens, McMahon and Podolsky (69) have recently reported that the inhibition by fluoroacetate salts of pyruvate-induced oxidations in yeast depends on the pH of the medium. However, inhibition of acetate-induced oxidations in yeast and *Chilomonas paramecium* is independent of pH. While this may have been one of the actual causes of differences in opinion concerning the specificity of fluoroacetate-induced inhibitions in yeast metabolism, Hutchens *et al.* find pyruvate oxidation much more sensitive than acetate oxidation in the case of *Chilomonas*. Although it is very hazardous to change species in the middle of an argument about fluoroacetate, it can be pointed out in support of this that Bueding (26) found that in the filarial worm, *Litomosoides carinii*, pyruvate oxidation is very much more sensitive than acetate. In addition, he stated, "No evidence has been obtained that fluoroacetate inhibits the respiration of the filariae because of a competitive inhibition of acetate oxidation." Fluoroacetate (0.001–0.004 M), while producing a decrease in the total respiration and motility of the organism, actually produced an accumulation of pyruvate and a decrease in the formation of acetate from glucose. Although in this study he has rigorously tested these

conclusions, Bueding also demonstrated that the metabolic characteristics of *L. carinii* are unique in that they differ from those of other helminths and, indeed, from those of most other invertebrates.

The anthropocentric may draw more comfort, therefore, from the results of a later study by Kalnitsky and Barron (78) on the effects of fluoroacetate and fluorobutyrate on fatty acid and glucose oxidation in kidney homogenates. Homogenates of rabbit kidney cortex oxidize acetic and many other fatty acids vigorously. The oxidation of acetate was immediately, and practically completely, inhibited by 0.001 M fluoroacetate and fluorobutyrate. This is in contrast to the total lack of effect of fluorobutyrate on acetate metabolism in yeast. This discrepancy was shown *not* to be the result of conversion of fluorobutyrate to fluoroacetate by kidney. Two other sharp differences between yeast and mammalian tissue were noted. The apparent release of the fluoroacetate-induced inhibition of yeast metabolism with time does not occur in kidney suspensions nor does ethanol have the least effect on the degree of acetate oxidation. Fluorobutyrate proved to be a more potent inhibitor of butyrate oxidation than did fluoroacetate; 0.00005 M fluorobutyrate inhibited butyrate oxidation 86% while at the same concentration fluoroacetate produced only 32% inhibition. Oxidation of higher fatty acids was also inhibited to varying extents by both fluoroacids.

In contradistinction to the case in yeast where it developed slowly, glucose oxidation was rapidly inhibited by fluoroacetate in kidney homogenates, as was also that of acetate. However, as in their previous studies, they found that pyruvate oxidation was not inhibited until a considerable portion (20%) of the added pyruvate had been oxidized. It appears that the specificity of fluoroacetate inhibition described for yeast is not to be found in mammalian tissue, in this case, rabbit kidney. It is interesting that fluorobutyrate is a more effective inhibitor of butyrate oxidation than is fluoroacetate. One can only speculate at this time about the effects of appropriately fluorinated higher fatty acids.

By actual analysis for acetate, Colowick, Berger, Slein and Cori (41) found that rabbit kidney cortex homogenates removed about 35% less acetate after addition of 0.005 M fluoroacetate. They add the new information that the extra oxygen consumption caused in dialyzed extracts of rabbit kidney cortex by addition of various metabolites is inhibited by high concentrations of fluoroacetate (0.05 M) to varying degrees. Glucose is most sensitive, the extra oxygen uptake being inhibited 68%, and phospho-enol pyruvate is approximately the same, the inhibition being 65%. Unfortunately, acetate does not appear to have been tested. The effect of fumarate was found to be inhibited 53% but other metabolites and components of the tricarboxylic acid cycle were relatively little affected. The curious sensitivity of fumarate-induced extra oxygen consumption does not appear to be entirely coincidental for Boyarski, Postel, Rosenblatt and Gerard (18) found it to be specifically effective in preventing the decrease in the action potential of methyl fluoroacetate-poisoned frog nerve. On the other hand, it actually increased the toxicity of sodium fluoroacetate to intact rabbits (36). These matters require considerable clarification. Liébecq and Peters (84) have reported recently that studies with centrifuged, homogenized guinea pig kidney and pigeon brain preparations indicate the possibility that a "fluoro-C_2 active fragment" may be formed which enters the tricarboxylic acid cycle and becomes an inhibitor of this cycle. They found, as did Kalnitsky (76), that citrate accumulates during poisoning *in vitro* while acetate does not. The relation of results of many *in vitro* studies to the events occurring *in vivo* is difficult to establish. It is possible that more physiological preparations could prove more useful.

B. Working Muscles

The use of an actively functioning, normally organized preparation such as the frog sartorius muscle has led to some additional and slightly different information. Colowick, Berger, Slein and Cori (41) found that the oxidative resynthesis of phosphocreatine by frog sartorius following one minute of tetanic stimulation was depressed as much as 40% by previously soaking the muscle for 1 hour in 0.005 M methyl fluoroacetate. These findings were explained as the indirect result of depressed tissue respiration, for the simultaneous oxygen uptake of these muscles following stimulation was decreased from an average 150% increase to only 35% increase. Essentially the same results were obtained when dinitrophenol was used to increase basal oxygen consumption, for 0.005 M sodium fluoroacetate inhibited the increase about 70%.

Similar results were obtained when caffeine was used by Clarke and Riker (39) to stimulate the frog sartorius, the excess oxygen consumption of muscles contracting under such circumstances being decreased by methyl fluoroacetate. The respiration of resting muscles is not affected significantly at similar concentrations of methyl fluoroacetate. The oxidative recovery heat which normally follows a single maximal twitch was abolished by 0.001 M methyl fluoroacetate or 0.01 M sodium fluoroacetate, yet the muscles continued to contract, exactly the reverse of the situation with iodoacetate where an increasing oxidative recovery heat production may accompany contractile failure. The depression of the activity oxygen consumption of these muscles induced by fluoroacetate can be abolished completely by 0.01 M acetate, glycerol monoacetate, pyruvate and oxalacetate. Ketoglutarate, fumarate, malate and succinate were less effective, and glucose and lactate were without any significant effect.

In a continuation of this study, Clarke and Riker have recently found that, unlike iodoacetate, fluoroacetate does not inhibit anaerobic glycolysis and, under the conditions of their experiments with frog muscle, the rate of glycolysis is actually increased. Because the aerobic accumulation of lactate that normally results from muscle activity is significantly less in the presence of fluoroacetate and since lactate formation is not inhibited by fluoroacetate, it appears that lactate must be metabolized in the poisoned muscle. The explanation of these results is to be found in the fact that, in this preparation at least, the action of fluoroacetate appears to be to inhibit oxidative carbohydrate breakdown; as a consequence, the anaerobic carbohydrate breakdown which occurs in activity is enhanced, and this results in a rapid turnover of lactate without its accumulation. It is evident that this will provide sufficient energy to permit muscular contraction.

Three independent studies of the effect of fluoroacetate upon the spontaneous contractility of isolated upper segments of rabbit small intestine have been made. The effectiveness of acetate as a source of energy for the contraction of otherwise substrate-free preparations of the type described by Furchgott and Shorr (55) led Furchgott to study the interrelations of fluoroacetate and acetate on this preparation. Experiments performed during 1946 by Furchgott (54) indicated that glucose could supply energy for contraction of intestinal smooth muscle in the presence of fluoroacetate under either aerobic or anaerobic conditions, although acetate could not. Fluoroacetate poisoning was irreversible when carried out under aerobic conditions. However, if fluoroacetate was added during a period of anoxia, allowed to remain in the muscle chamber for over 30 minutes and then washed out before the restoration of oxygen to the muscle, there was no toxic effect.

Later, Farah, West and Angel (50), upon examining this system found that both glucose and acetate were effective antagonists to fluoroacetate, and that there are considerable differences in the character of the response of the gut to fluoroacetate in the presence of these substrates. They have shown that although fluoroacetate depresses contractility more rapidly when glucose is the sole substrate than when acetate alone is present, the percentile decrease in amplitude of contraction at a given concentration of fluoroacetate is greater in the presence of acetate than when glucose is the substrate. There is a direct relation between (a) the time required for 0.0008 M sodium fluoroacetate to produce 95 to 100% inhibition of contraction and (b) the concentration of sodium acetate present in the bath, the higher the concentration of acetate the longer the time required for this inhibition to result. Butyrate and pyruvate, when they are the sole substrates, are not detectably different from acetate with respect to the percentile reduction in contraction amplitude produced by a given concentration of fluoroacetate.

Although regular contractions in glucose are stopped by high concentrations of fluoroacetate, there remains an irregular, high amplitude "fluoroacetate resistant" contraction when glucose is present with or without other substrates; this phenomenon is seldom or never seen when acetate alone is the substrate. It was noted by all groups which investigated the problem. Farah *et al.* (50) have found that only mannose acts like glucose whereas galactose, fructose, pyruvate, butyrate, caproate, caprylate, succinate, fumarate and α-keto-glutarate are, like acetate, unable to support these contractions. These contractions are not abolished by anaerobiosis (N_2, cyanide) or by malonate, although azide and iodoacetate are effective in abolishing them. It has been known for some time, as Farah *et al.* have pointed out, that intestinal smooth muscle can utilize glucose and mannose as a source of contraction energy during anaerobiosis. They feel that it is possible that energy for the fluoroacetate resistant contractions may be obtained from anaerobic glycolytic pathways.

That glucose is more effective than acetate in maintaining motility of gut segments in the presence of high concentrations of fluoroacetate was confirmed by Weeks and Chenoweth (132). When intestinal strips are allowed to contract after exhaustion in Krebs-Henseleit solution in the absence of substrate, the normal stimulatory action of added 0.005 M sodium acetate is prevented by addition of 0.00032 M sodium fluoroacetate 5 minutes before the sodium acetate, whereas subsequent addition of 0.005 M glucose is still effective. Although they found that glycerol monoacetate is a very effective antagonist to fluoroacetate *in vivo* and sodium acetate is definitely not, in the isolated intestinal segment preparation sodium acetate is nearly five times as effective as glycerol monoacetate against fluoroacetate. Other experiments demonstrated that glucose-induced contractions, after fluoroacetate inhibition (0.01 M) in acetate, were of the same amplitude (approximately 30% of control values) as the contractions which persisted when the same concentration of fluoroacetate was added to muscles with a glucose substrate. These various observations can probably be explained by assuming, and there appears to be good reason to do so, that intestinal muscle under these circumstances obtains energy for contraction from at least two sources: (1) the breakdown of glucose which can occur anaerobically and (2) reactions of the tricarboxylic acid cycle into which acetate enters. When acetate, as the sole available substrate, is blocked from entry into the cycle by high concentrations of fluoroacetate, contraction ceases; in contrast, low concentrations of fluoroacetate are unable to produce a block in the presence of excess acetate. In the presence of glucose, energy for contraction is still available despite the blockade of acetate produced by fluoroacetate. This blockade may be increased until only the fluoroacetate, anaerobiosis-resistant contractions remain.

C. Working Nervous Tissue

According to Shanes and Brown (117), the preservation of the resting potential of frog nerve depends upon formation of pyruvate by the glycolytic cycle and the subsequent aerobic metabolism of this substrate. Because 0.01 M methyl fluoroacetate interferes with the redevelopment of the resting potential of nerve in oxygen following a period of anoxia, Shanes (116) felt that this was sufficient to suggest an interference with pyruvate metabolism. Conversely, the simultaneous addition of methyl fluoroacetate and sodium pyruvate to nerve in oxygen maintains a higher resting potential than when pyruvate is omitted, a fact which suggests a beneficial effect of added pyruvate not noted with acetate. During poisoning, he found that the threshold of excitability of frog sciatic nerve to condenser discharges steadily increased.

A series of studies has recently been published which has revealed several new facts about the action of fluoroacetate (17, 18, 22, 23, 45, 98). The sodium salt of fluoroacetic acid is nearly without action on frog nerve or brain *in vitro* although methyl fluoroacetate has several actions. Thus, the ester decreases the action potential of frog sciatic nerve and reduces conduction velocity by a process of blocking conduction in fibers; the threshold of the larger fibers is raised before that of the smaller fibers at concentrations of 0.005 M. The respiration of such nerves is decreased to 20% of normal. Sodium fumarate added before, or up to 15 minutes after, the methyl fluoroacetate protects against the action

potential changes in a 2:1 molar ratio, but the respiration of the nerve may still be 50% decreased. Succinate is equally effective in a 5:1 molar ratio; ethanol, acetate, pyruvate, α-keto-glutarate and glucose are ineffective (1). Although their data have not yet been fully reported, Doty and Gerard (5) have found that methyl fluoroacetate will depress the resting oxygen consumption of frog nerve as described, but that the increased oxygen consumption which occurs on stimulation is not affected. Thus, with 0.001 M methyl fluoroacetate the resting Q_{O_2} is decreased by 25% whereas the activity increase is unaffected and the action potential is undisturbed. They caution, however, that the presence of respiring, non-conducting elements in a nerve trunk must be considered when interpreting these results.

In Gerard's isolated frog brain preparation, sodium fluoroacetate is ineffective whereas methyl fluoroacetate inhibits respiration 45% at 0.012 M (23). At this level the brain potentials are decreased about 50%. At 0.01 M methyl fluoroacetate there may also be a 50% decrease in cholinesterase activity. On this preparation the beneficial effects of fumarate are again seen, but they are rendered somewhat less specific by observations that fumarate protects to some extent against di-isopropyl fluorophosphate and that under certain circumstances as little as 0.000,001 M sodium fumarate can itself induce bizarre electrical changes in the brain.

The inactivity of sodium fluoroacetate does not appear to be solely the result of inability to penetrate cells, for it is inactive on rat brain or dog nerve homogenates in which cells are disrupted. Malic dehydrogenase of rat brain is sensitive to methyl fluoroacetate (20% inhibited at 0.001 M), but in general dehydrogenases are not much affected by fluoroacetate (98).

D. *Isolated Perfused Hearts*

By the use of a recirculating system for perfusing isolated hearts through the coronary arteries over long periods with bacteria-free Ringer's solutions (32) it has been found that concentrations of methyl or sodium fluoroacetate comparable to those calculated to exist in rabbits or rhesus monkeys poisoned with LD_{100} cause a gradual decline in the amplitude of contraction, occasional tetanans and rarely, if ever, fibrillation (28). The relative sensitivity of rabbits and monkeys to fluoroacetate is also manifested in their isolated hearts. Essentially the same degree of cardiac incompetence was produced by methyl fluoroacetate acting over a two-hour period in a concentration of 0.00001 m (0.5 mgm./L) on the isolated rabbit heart as by an intravenous dose of 0.5 mgm./kgm. to the intact animal (equivalent to about 0.6 mgm./L of body water). In the case of the monkey heart, 0.001 M fluoroacetate in the perfusion fluid produced effects which correspond roughly to those of an intravenous dose of 5 to 10 mgm./kgm.

The substitution of sodium acetate for the glucose of the perfusate in the rabbit and monkey hearts effectively maintained contraction and exerted an extensive protection against the effect of added fluoroacetate (33). In some instances protection was definite when the molar ratio of acetate to fluoroacetate approximated unity, but usually a higher ratio was necessary. Pyruvate was somewhat less effective in maintaining contractions and in protecting against fluoroacetate, perhaps only because of toxic impurities (20).

Because the presence of acetate prevented the action of practical concentrations of fluoroacetate, it was impossible to demonstrate any change in the utilization of acetate by the heart in the presence of fluoroacetate. However, when failure was produced by the addition of 0.01 M fluoroacetate to monkey hearts with either glucose or pyruvate as the substrate, there was an acetate accumulation of as much as 3 grams/kgm. dry weight/hour over the three-hour exposure period. No other change in the utilization or production of acetate, pyruvate, citrate, α-keto-glutarate or glucose was detected.

Despite the protection exerted *in vitro*, sodium acetate by itself exerts no protective effect whatever against the toxic action of fluoroacetate in the intact rabbit (36, 37).

E. *Mechanism Studies in Intact Animals; Antidotes*

Because humans accidentally or wilfully ingest rat poisons, there can be no doubt of the desirability of an effective antidote to fluoroacetate. The studies so far described do not offer much hope that any highly effective treatment of well-established fluoroacetate poisoning will be found. Indeed, most investigators have been content if their results with antidotal therapy contribute something to an understanding of the mechanism of action of the poison.

It has been mentioned that sodium acetate, although it is the most likely candidate, is not an effective antidote or prophylactic in rabbits poisoned with fluoroacetate. However, Tourtelotte and Coon (125) have found that in mice, at least, sodium acetate (2 to 3 gram/kgm.) will protect against sodium fluoroacetate. Ethanol, which may be considered simply a source of acetate *in vivo* or, more complexly, a catalyst of the Krebs' cycle, is also effective (1.6 gram/kgm.). Ethanol and acetate together are distinctly more than twice as effective as either alone, a fact which suggests a synergistic effect.

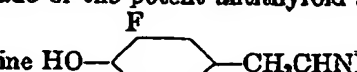
Hutchens *et al.* (70) have reported more fully on the effectiveness of ethanol alone or with sodium acetate in protecting mice against fluoroacetate. Ethanol is also effective in rabbits, and to a lesser extent in guinea pigs, but not at all in dogs. It was noted that barbiturates were somewhat effective in protecting dogs but not mice. Although there is no doubt that the judicious administration of barbiturates (bearing in mind the prolongation of sleeping time described in III-G, c.) will control convulsions induced by fluoroacetate in dogs (52, 70, 92), animals which survived in this laboratory manifested changes characteristic of cortical damage. It would thus appear that, although overt convulsions are prevented, the pathological pattern of fluoroacetate poisoning has been unaffected.

The effectiveness of sodium acetate *in vitro*, despite a generally unfavorable response to it *in vivo*, led to a search for other sources of C_2 moieties. One of the most promising has been glycerol monoacetate which has protected rats, rabbits, dogs and rhesus monkeys against fluoroacetate (36, 37). Equimolar doses of

glycerol monoacetate and ethanol, given to rhesus monkeys after fluoroacetate poisoning had progressed, were strikingly different in effectiveness, ethanol apparently being of no value although its effectiveness in mice and rabbits has been confirmed. Several other such compounds have been found which exert protective action against either fluoroacetate or fluorocrotonate.

The simultaneous administration of large amounts of insulin and glucose is often effective in dogs and rabbits, although neither substance is effective alone (37). (The forcing of glucose is also protective against anoxic anoxia (2).) Anoxia caused by methemoglobinemia (approximately 70%) produced either by sodium nitrite or p-aminoacetophenone is a very effective antidote or prophylactic for fluoroacetate poisoning in those species in which it can be induced, *e.g.*, mice and dogs (37). McNamara (97) has recently found that physiological sodium chloride solution exerts a definite protective effect against fluoroacetate poisoning in rabbits, a fact which may account, in part at least, for the heterogeneous character of some of the substances that appear to exert a moderate protective action. It serves no useful purpose to list in detail all those substances which have proven ineffective; but, in general, salts of fatty acids, anticonvulsants, vitamins and most metabolic intermediates are without effect. Potent antifibrillatory, autonomic and cardiac drugs are generally of no therapeutic value; they may act differently after fluoroacetate (e.g., 50).

V. MISCELLANEOUS FLUORINATED COMPOUNDS

Several familiar compounds in which fluorine has replaced hydrogen or chlorine have been prepared and are of some interest here. Although 2,3-difluoro succinic acid (80), HOOC—CHF—CHF—COOH, appears to inhibit succinic dehydrogenase completely in very low concentrations, it is of singularly low toxicity, the LD_{50} in mice and dogs (salt or dimethyl ester) being above 200 mgm./kgm. (37). When fluorine is substituted for chlorine in sesqui-H, a very potent analog of mustard gas, the resulting compound $F—CH_2CH_2—S—CH_2CH_2—S—CH_2CH_2—F$, is nontoxic with neither vesicant nor fluoroacetate-like activity (95). This suggests that the body is unable to rupture the C-S link in this compound to obtain fluoroacetate and adds emphasis to the fact that the vesicant action of the mustards is dependent upon reactive halogens. Other compounds in which fluorine has been substituted for an hydrogen atom, such as di- (2-fluoroethyl) fluorophosphate or triethyl lead fluoroacetate, combine some of the characteristic activity of the parent compound with that of fluoroacetate (109). Mention should be made of the potent antithyroid activity reported by Litzka (87) for 3-fluorotyrosine  . This activity may be because of the high toxicity (LD_{50}, 12.5 mgm./kgm.), since 1- or 4-fluorobenzoic acids are virtually nontoxic (79, 86). Boyer, Evans and Phillips (19) were unable to demonstrate an effect on the basal metabolic rate of rats, although they confirmed the toxicity of the compound. These compounds have been included because they give some indication of the way in which fluorine atoms in place

hydrogen atoms may be used to obtain information concerning biological reactions.

VI. DISCUSSION

Nearly all the original data on monofluorinated fatty acids so far available in the open literature and much of that in the classified literature have been presented in this review. There appears to be a plethora of information on less important points but a dearth of incontrovertible data on the more important questions. What interpretation can be given the facts now available?

It is clear that the substitution of one fluorine atom for one hydrogen atom in a compound which is known, or suspected, to be of biological importance confers unusual pharmacological properties upon that compound provided that the substitution be made in the proper place. This proper place is always in the terminal position of a straight chain fatty acid containing an even number of carbon atoms, to cite the best-defined series. Thus 2-monofluoroacetic acid is active, 3-monofluoropropionic acid is inactive and 4-monofluorobutyric acid is very active, although 2-monofluorobutyric acid is inactive, and so on. Substitution of any atoms or groups other than one fluorine atom on the terminal carbon atom does not result in characteristic activity. More complex compounds containing a suitable grouping, for example, esters of 2-monofluoroacetic acid or 2-fluoroethanol, exert a typical pharmacological action if they can be broken down in the organism to yield fluoroacetic acid. This principle has been applied in a few less simple cases to indicate whether or not the body can rupture certain linkages, the C-S bonds in the chain F—C—C—S—C—C—S—C—C—F being an example. It appears to be a technic of some value, although open to criticism on the grounds that such a compound might be a specific inhibitor *per se*, should it not prove to be inert (for instance, fluoroacetyl salicylic acid).

The toxicity of suitably substituted even-numbered fatty acids in contrast to the inertness of odd-numbered homologs is superficially further evidence for the oxidation of fatty acids, the assumption being that the even-numbered chains are broken down to the toxic fluoroacetic acid. Then, in order to account for the much greater toxicity (and the relative differences in various species) of the longer chains as compared to fluoroacetic acid on a mole per kilogram basis, it is necessary to *assume* that (a) they penetrate cells much more efficiently and/or (b) that release of fluoroacetate as an "active" $F\text{-}C_2$ radical occurs, preferably directly in the area where it could do the most harm. To the contrary, the quantitative and qualitative differences between the actions of the fluorinated higher fatty acids and the shortest active acid suggest most strongly that they act at separate sites. Evidence has been presented that fluorobutyrate, for example, is a much more active inhibitor of butyrate oxidation in mammalian tissue than is fluoroacetate, while in yeast fluorobutyrate does not inhibit acetate oxidation at all although fluoroacetate actively does so. Under at least one set of circumstances it has been shown that fluorobutyrate does not break down to fluoroacetate. In addition, the gross pharmacological effects of fluorobutyrate differ qualitatively from those of fluoroacetate, although admittedly the difference requires

sharper definition. While it is impossible on the basis of available inform[illegible] to exclude the possibility of β-oxidation of fluorinated higher fatty aci[illegible] fluoroacetate, it appears probable that they exert their toxic action in pa[illegible] least, by interfering with the metabolism of the corresponding non-fluori[illegible] fatty acid in the cell. It is thus equally easy to dispose of the inactivity o[illegible] numbered fluoroacids, for it is generally uncommon to find the non-fluori[illegible] homolog important in the main line of fatty acid oxidation.[4]

Certain inactive compounds shown in Table III might be used to [illegible] weight to the hypothesis of β-oxidation to fluoroacetate. For example, F—CH[illegible]

$$\mathrm{CH_3}$$
$$|$$
$\mathrm{C—CH_2COOH}$ is pharmacologically inactive, and on structural grounds [illegible]
$$|$$
$$\mathrm{CH_3}$$

not be expected to undergo β-oxidation to fluoroacetic acid. Similarly, deriva[illegible] of fluorobutyric acid in which carbon atoms 2 and 3 are part of a cyclic stru[illegible] are inactive, and are not susceptible to β-oxidation. Although the inactivi[illegible] these compounds adds support to the idea that β-oxidation of higher ω-flu[illegible] acids is necessary for activity, it is equally possible to argue that becau[illegible] steric factors these biologically abnormal compounds never have an opportu[illegible] to inhibit butyrate oxidation inasmuch as they are kept from entering a rea[illegible] center by their structural deformity and for that reason are pharmacologi[illegible] inert.

In this case the assumption of "entrance into reactive centers" is based en[illegible] upon the remarkable similarity (see Table I) of the FCH_2- and HCH_2- radi[illegible] the chief differences being that the fluorine atom is about twice the size of [illegible] hydrogen atom and is bound more securely to the carbon atom. (Were ther[illegible] physical-chemical differences at all there would seem to be no reason to ex[illegible] any pharmacological differences.) If it be assumed that in the process of me[illegible] olism fatty acids fit some sort of matrix which requires entry of the termi[illegible] methyl group, it is easy to visualize the arrival of a fluorinated methyl g[illegible] which fits effectively; but, perhaps because of the greater hold the carbon [illegible] fluorine atoms have upon each other and because of fluorine's propensity [illegible] hydrogen bonding, it cannot readily be dislodged. In such a case as fluoroa[illegible] acid the prior administration of substances which release large amounts of a[illegible] acid in the cell could be expected to influence this phenomenon by compe[illegible] for the fixation site. Poisoning by fluoroacetate can indeed by prevented or [illegible] reversed by such substances in intact animals and in some isolated syst[illegible] Unfortunately, a different interpretation can be placed upon this observa[illegible] There is no way of knowing with certainty whether added acetate comp[illegible] with fluoroacetate or simply by-passes a blockade. To clarify this it is nec[illegible]

[4] Inactivity may be a relative matter because the doses of odd-numbered compo[illegible] tested do not seem to have exceeded 200 mgm./kgm. and have been examined only in [illegible] species. It is possible that some organisms may be very sensitive if odd-numbered [illegible] acids are actively metabolized by them.

[illegible] marshal the data concerning the various possible blockades which have been [illegible]ested.

[illegible] Although it would be extremely helpful to have more data on other com[illegible]unds, nearly all the detailed investigations concerning inhibition of metabolic [illegible]cesses by the fluoroacids have been performed with fluoroacetic or fluoro[illegible]yric acid. The specificity (or even the existence) of the inhibition of the [illegible]dation of acetate and pyruvate by fluoroacetate has been the chief subject [illegible]tudy, the results of which have been vastly complicated by differences in [illegible]nic, species or organs employed. (One searches the literature in vain for [illegible]ntion of the existence of fluoropyruvic acid, $F—CH_2—\underset{\underset{O}{\|}}{C}—COOH$, which [illegible]ht be utilized to settle some of these questions.)

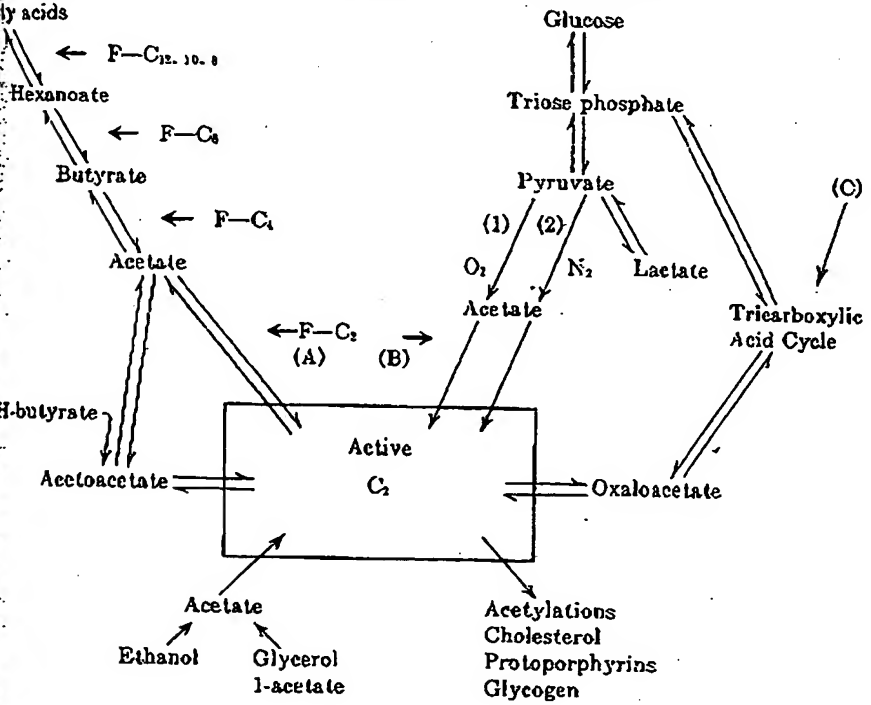
[illegible]t is fairly certain that fluoroacetic acid does not inhibit acetylation of foreign [illegible]atural amines by mammals. The formation of acetoacetate from acetate is [illegible] inhibited and in general—*Litomosoides carinii* being a clear exception—the [illegible]dation of pyruvate to acetate may be directly affected while the disposition [illegible] the acetate so formed is certainly affected. Acetate probably exerts an in[illegible]bitory effect upon pyruvate oxidation after a sufficient amount of it has [illegible]umulated. There appears to be no way at this time of choosing between [illegible]dings which suggest inhibition of fumarate oxidation and those which do not. [illegible] this connection it is possible to argue obliquely that, because fluorosuccinate [illegible] other potent competitive inhibitors of succinic dehydrogenase are relatively [illegible]ble animal poisons, inhibition of other neighboring steps in the Krebs' cycle [illegible]ot likely to be the cause of death in mammals poisoned with the usual minute [illegible] of fluoroacetate. The effects on citrate and glucose metabolism may best [illegible] explained as the indirect result of inhibition at other points.

[illegible]t is well established that the breakdown of glucose to triose phosphate can [illegible]ur anaerobically and aerobically, and is a source of much energy. Pyruvate [illegible] long been known as the main end-product of this metabolic chain, lactate [illegible]g a simple conversion product of pyruvate. However, in the last decade the [illegible]fragment, a much discussed entity having many but not all of the character[illegible]ics of acetate, has been definitely added as a stage of carbohydrate oxidation [illegible]ond pyruvate and as the end-product of fatty acid breakdown (16, 83, 121). [illegible]fore presenting the schema depicted in Figure 1, it is necessary to bring out [illegible]ain less well known observations which relate to the possibility of its general [illegible]licability and to the action of fluoroacetate. Differences among species with [illegible]ard to the action of fluoroacetate must be more than a matter of vagary. If [illegible] inhibitions of various metabolic systems and pathways and the responses [illegible] intact animals which have been reviewed here are considered in the light of [illegible] importance of these systems and pathways to the economy of the organism [illegible]g studied, some interesting relations are obtained. Thus, although there are [illegible] qualitative differences between Wistar and Sprague-Dawley strains of rats [illegible] the way they handle the acetate in their acetate pool, Wistar rats turn over [illegible] acetate (12 to 15 mM/100 gram/24 hr.) than do Sprague-Dawley rats (20

to 25 mM/100 gram/24 hr.). In addition Wistar rats are unable to convert py... ruvate to acetate although Sprague-Dawley rats do so freely (4). The differenc... in response to fluoroacetate is clear; the strain to which acetate is more impo... tant is more sensitive to fluoroacetate (although more resistant to pyridoxin... deficiency (27) or alloxan (82)). It has recently been shown that intact rabbit... metabolize formate actively, but that dogs and man are nearly unable to do so; incidentally, this accounts for the relative toxicity of methanol to these two latter species (88, 89, 90). Dog muscle, on the other hand, can oxidize β-hydroxy... butryate and acetoacetate whereas rabbit muscle can only use acetoacetate (124). The differences between dogs and rabbits in response to fluoroacids are striking. Perhaps a similar relation exists when still other species are considered... for isolated mammae of the goat, a fluoroacetate-sensitive animal, utilize acetate whereas those of the relatively resistant rat and still more resistant mouse do not appear to do so (51). Mention has been made of the ability of guinea pig brain to oxidize added acetate and the relative inability of rabbit brain to do so, the fluoroacetate-induced inhibition of acetate oxidation being greater in brain tissue from the guinea pig than from the rabbit. The convulsive pattern of fluoroacetate poisoning in the guinea pig and the effects on the heart of the rabbit, which oxidizes acetate vigorously (10, 11, 33), intimate that this is no mere coincidence.

When one views these scattered observations on the relation between fluoroacetate action and metabolic pathways in the light of the protective effect of acetate and its donors, it is not unreasonable to suspect that the degree of sensitivity of an animal or its organs to fluoroacetate is an indication of a certain characteristic of its acetate metabolism. To establish this suspicion as a fact will require much effort and it is probable that the problem is even more complicated than appears at first. Reference has been made to studies with isolated frog muscle, rabbit intestine and intact animals which show that there are fluoroacetate-insensitive metabolic pathways giving rise to sufficient energy to maintain function. Evidence has been adduced to indicate that the breakdown of glucose is not sensitive to fluoroacetate and that some such system can be enhanced *in vivo* by partial anoxia (methemoglobinemia) or by accelerating the breakdown with an excess of glucose and insulin. It is highly probable that in addition to individual peculiarities of the metabolic systems for handling C_1-C_4 molecules in a given species a second factor, the glycolytic rate, is important in controlling the sensitivity of the organ or organism. Few comparative data have been published although Wu and Chang (134) have recently pointed out that the glycolytic rate of isolated eel, toad, turtle and rat hearts decreased (in that order) with their resistance to anoxia. Grossly, the sensitivity to fluoroacetate likewise decreases in the direction of greater glycolytic activity. It is also well known that very young mammals are very much more resistant to anoxia than are older ones. Nine 24-hour old dogs recently tested in this laboratory were markedly resistant to fluoroacetate. Further, the relative sensitivity of various vertebrates to tissue anoxia induced by potassium cyanide (67) is strikingly similar to their order of sensitivity to fluoroacetate. Farah (50) has called attention to the similarity between the sensitivities to fluoroacetate, cyanide and anoxia of various portions of the gut, which follow Alvarez's concept of a metabolic gradient. Although it is surprisingly difficult to find accurate comparative data (64), such adult mammals as have been found very resistant to anoxia (21) are also resistant to fluoroacetate.

FIG. 1. Tentative Localization of Fluoroacid Blockades



...action (1): Oxidative decarboxylation of pyruvate to acetate.
...action (2): Anaerobic degradations of pyruvate to acetate and lactate. Enhanced by partial anaerobiosis *in vivo* (methemoglobinemia) and by large amounts of glucose, thus minimizing effect of block (B).
...blockade (A), (B): Competitively antagonized by C_1 donors.
...blockade (C) and others not shown: Minor blockades caused by imperfect introduction of F—C—C—R in high concentrations into a matrix designed for other structures, *e.g.*, malic dehydrogenase.

In Figure 1, the anaerobic degradation of pyruvate to acetate by coupled oxidation-reduction reactions ("diamutation") to lactate and acetate, or by fermentation ("dissimilation") to acetate and formate, or by still other processes, is shown as unaffected by fluoroacetate, although oxidation of pyruvate and fatty acids through acetate to active acetate is shown as blocked. If allowance is made for variations among species and strains of organisms, such a blockade can account for many of the pharmacological responses so far described. One

phenomenon, however, is outstandingly unexplained. If blockades by the hig
fluoroacids actually occur as indicated, why are these acids so much more to
Perhaps this will become clear as the understanding of fatty acid metabo
increases. On the whole, it seems possible to explain the pharmacological ac
of fluoroacetate itself, together with some of the specific variations noted,
function of the magnitude and character of the acetate and glycolytic meta
pathways. The characteristic response of an organism or tissue to fluoroace
may be determined by the relative importance of these two pathways.

SUMMARY

Conversion of a metabolic intermediate into a very highly toxic compou
by the introduction of a single fluorine atom in a strategic position in the mole
has been described for a number of compounds. It appears to be a useful meth
for producing agents with which metabolic pathways can be differentiated i
large number of species with a minimum of effort, for it is evident that th
agents act by virtue of their close resemblance to natural metabolites. As
example, the variation among species in response to monofluoroacetic acid
been related to certain definite differences in metabolism in the species studi

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aided greatly in obtaining declassification.

ADDENDUM

The gracious permission of the British Ministry of Supply to refer to inform
tion in certain of their classified reports was received too late to permit inclusi
of this information in the body of the review. Mention should be made, howev
of the high resistance of the *Cercopithecus*, or "green" monkey to methyl fluor
acetate. The LD_{50} appears to lie above 50 mgm./kgm. according to K. J. C
penter and B. A. Kilby (1944). These workers also found that although fluo
ethanol is as active as fluoroacetate in intact animals, it is without action on t
isolated perfused heart, presumably because this organ can not convert fluor
ethanol to fluoroacetate.

Definite histologic abnormalities in the myocardium, but no unequivoc
changes in the central nervous system, were reported by A. M. Barrett (194
in guinea pigs and rabbits poisoned repeatedly with methyl fluoroacetate. T
beating of heart muscle cells in culture is rapidly inhibited by methyl fluoroac
tate but the growth of the cell masses is not affected, according to C. B. All
and H. B. Fell (1944). The action is specific, for methyl chloroacetate in equi
lent concentrations merely kills the cells.

The view that fluoroacetic acid is metabolized to fluorocitric acid, accounti
for inhibition of citrate oxidation and the subsequent accumulation of cit
acid, has been advanced by Martius (Ann. d. Chem. *661*: 227–232, 1949). Th
would seem referable to Blockade (C) of Figure 1.

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THE EFFECTS OF DRUGS UPON THE ELECTRICAL ACTIVITY OF THE BRAIN*

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I. INTRODUCTION

It is now twenty years since Hans Berger (20) presented to the world a revolutionary new method for the investigation of the function of the brain in health and disease, namely, *electroencephalography*. His contribution was threefold. To the clinician he gave a diagnostic instrument with sharp differential and localizing value, without which the notable advances of the last two decades in the pharmacological and surgical treatment of central nervous disorders would have been sorely handicapped. To the neurophysiologist he gave a research tool of great precision and relative simplicity for the unraveling of the central nervous pathways by the monitoring of their electrical signs. Lastly, to all those who strive to understand the mode of operation of the organ of thought, he gave a new point of departure. The brain could no longer be considered a passive switchboard through which impulses coursed on their way to and from the periphery; it was now revealed as a dynamic participant in the affairs of the body, possessing an inherent spontaneous and rhythmic activity which could modify the soma and be modified in turn by the soma.

Berger did not neglect to investigate the effects of drugs upon the electrical activity of the brain (21). He looked for electroencephalographic signs of the central nervous actions of barbital, morphine, cocaine, amyl nitrite, scopolamine, chloroform and other substances then in common clinical use. Thus he opened a field of research which has many potentialities not only for the determination of the mechanism of action of centrally acting drugs, but also for the analysis of the nature of the electroencephalogram itself, for the reason that substances with known specific action are among the most precise tools available for investigations in the biological sciences.

Unfortunately, the rapidly increasing volume of literature in the field of electroencephalography contains relatively few systematic studies of the effects of drugs on the electrical activity of the brain, although there are many empirical reports on the alterations produced by particular chemical agents. Among previous reviews relevant to this subject should be mentioned those of Hayslip (176), Gibbs and Gibbs (135), Finesinger and Brazier (108), Gibbs (130, 131, 132), Lennox and Lennox (217), and Walter and Walter (331).

The present review will consider primarily the effects of drugs upon the normal human EEG. This will necessarily exclude from discussion most of the large

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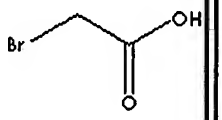
BROMOACETIC ACID



PRODUCT IDENTIFICATION	
CAS NO.	79-08-3
EINECS NO.	201-175-8
FORMULA	$BrCH_2COOH$
MOL WT.	138.95
H.S. CODE	
TOXICITY	Oral rat LD50: 50 mg/kg
SYNONYMS	2-Bromo acetic acid; alpha-Bromoacetic acid; Monobromoacetic acid;
DERIVATION	
CLASSIFICATION	
PHYSICAL AND CHEMICAL PROPERTIES	
PHYSICAL STATE	white to light yellow crystalline powder
MELTING POINT	49 - 51 C
BOILING POINT	206 - 208 C
SPECIFIC GRAVITY	1.934
SOLUBILITY IN WATER	soluble
pH	
VAPOR DENSITY	
AUTOIGNITION	
NFPA RATINGS	Health: 3; Flammability: 1; Reactivity: 0
REFRACTIVE INDEX	
FLASH POINT	
STABILITY	Stable at ordinary conditions. Hydroscopic.

APPLICATIONS

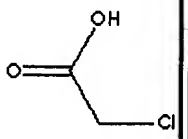
Bromoacetic Acid is used as a chemical intermediate for the manufacturing other compounds and pharmaceuticlas.

SALES SPECIFICATION	
APPEARANCE	white to light yellow crystalline powder
PURITY	98.0% min
TRANSPORTATION	
PACKING	
HAZARD CLASS	3 (Packing Group: II)
UN NO.	1938

OTHER INFORMATION

Hazard Symbols: T C, Risk Phrases: 23/24/25-36, Safety Phrases: 36/37/39-45

CHLOROACETIC ACID



PRODUCT IDENTIFICATION

CAS NO.	79-11-8
EINECS NO.	201-178-4
FORMULA	$CH_2ClCOOH$
MOL WT.	94.50
H.S. CODE	
TOXICITY	
SYNONYMS	Chloroethanoic acid; Monochloroethanoic acid; ácido cloroacético (Spanish); Acide chloroacétique (French); alpha-Chloroacetic acid; MCA; Monochloroacetic Acid; Monochloorazijnzuur; Monochloressigsäure (German); Acidomonochloroacetico (Italian);
DERIVATION	acetic acid , chlorine
CLASSIFICATION	

PHYSICAL AND CHEMICAL PROPERTIES

PHYSICAL STATE	white solid
MELTING POINT	61 - 62 C
BOILING POINT	188 C (solid), 143 C (80% aqueous solution)
SPECIFIC GRAVITY	1.40 (solid), (1.20 80% aqueous solution)
SOLUBILITY IN WATER	Soluble
SOLVENT SOLUBILITY	Soluble in ether, chloroform, benzene, and alcohol
pH	<1 (80% aqueous solution)
VAPOR DENSITY	
AUTOIGNITION	
NFPA RATINGS	Health: 3; Flammability: 1; Reactivity: 0
REFRACTIVE INDEX	
FLASH POINT	126 C (solid), (1.20 80% aqueous solution)
STABILITY	Stable under ordinary conditions. Hygroscopic.

DESCRIPTION AND APPLICATIONS

Monochloroacetic acid, chlorinated simplest carboxylic acid, has electron-withdrawing atom (alpha-chlorine) on the next carbon to acid. Alpha-chlorine makes monochloroacetic acid more acidic than acetic acid. It is used as one of the first choice chemical intermediates for the production of;

- Carboxy Methyl Cellulose and Starch
- Phenoxyacetic Acid
- Thioglycolic Acid
- Cyanoacetic Acid / Malonates / Barbituric Acid
- Caffeine, Betaine, Vitamin B and pharmaceuticals
- Glycine
- Surfactants
- Indigo dyes
- Herbicides

SALES SPECIFICATION

APPEARANCE	white flakes
ASSAY	99.0% min

THE MERCK INDEX

AN ENCYCLOPEDIA OF
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USE: In water disinfection; bleaching fibers and silk; manuf medicinal bromine compds, dyestuffs.

1416. Bromine Pentafluoride. BrF_5; mol wt 174.90. Br 45.69%, F 54.31%. Prepd by fluorination of bromine at 200° in iron or Monel metal apparatus: Ruff, Menzel, *Z. Anorg. Allgem. Chem.* **202**, 49 (1931); Kwasnik in *Handbook of Preparative Inorganic Chemistry*, Vol. 1, G. Brauer, Ed. (Academic Press, New York, 2nd ed., 1963) pp 158-159; from F_2 and KBr: Hyde, Boudakian, *Inorg. Chem.* **7**, 2648 (1968). *Reviews:* Kemmitt, Sharp, *Advan. Fluorine Chem.* **4**, 243-244 (1965); Stein, "Physical and Chemical Properties of Halogen Fluorides" in *Halogen Chemistry*, Vol. 1, V. Gutmann, Ed. (Academic Press, New York, 1967) pp 133-224; Meinert, *Z. Chem.* **7**, 41-57 (1967).

Liquid. Fumes in air. mp −60.5°. bp 40.76°. d^{25} 2.4604. Trouton constant 23.7. Thermostable up to 460°. Does not attack quartz when dry. *Produces an explosion on contact with water.* Very reactive, usually with conflagration.

Caution: Corrosive and irritating to eyes, skin, mucous membranes.

1417. Bromine Trifluoride. BrF_3; mol wt 136.90. Br 58.37%, F 41.63%. Prepd by fluorination of bromine at 80°: Lebeau, *Compt. Rend.* **141**, 1018 (1905); Prideaux, *J. Chem. Soc.* **89**, 316 (1906); Ruff, Braida, *Z. Anorg. Allgem. Chem.* **206**, 59 (1932); **214**, 91 (1933); Simons, *Inorg. Syn.* **3**, 184 (1950); Kwasnik in *Handbook of Preparative Inorganic Chemistry*, Vol. 1, G. Brauer, Ed. (Academic Press, 2nd ed., 1963) pp 156-157. *Reviews:* Kemmitt, Sharp, *Advan. Fluorine Chem.* **4**, 244-245 (1965); Stein, "Physical and Chemical Properties of Halogen Fluorides" in *Halogen Chemistry*, Vol. 1, V. Gutmann, Ed. (Academic Press, New York, 1967) pp 133-224; Meinert, *Z. Chem.* **7**, 41-57 (1967).

Colorless liquid; also reported to be pale yellow. Long prisms when solid. mp 8.77°. bp 125.75°. d^{25} 2.8030. *Smokes in air. Attacks skin. Very reactive.*

Caution: Corrosive and irritating to skin, eyes, mucous membranes, respiratory tract.

USE: Solvent for fluorides.

1418. Bromisovalum. *N-(Aminocarbonyl)-2-bromo-3-methylbutanamide; (α-bromoisovaleryl)urea;* 2-monobromoisovalerylurea; α-bromo-β-dimethylpropanoylurea; bromalcetone; B.V.U.; Bromural; Bromisoval; Uvaleral; Bromuran; Somnurol; Brovalurea; Dormigene; Isobromyl; Alluval; Vivadorm. $C_6H_{11}BrN_2O_2$; mol wt 223.07. C 32.31%, H 4.97%, Br 35.82%, N 12.56%, O 14.34%. $(CH_3)_2CHCHBrCONHCONH_2$. Prepn from urea and α-bromoisovaleryl bromide: U.S. pat. **914,518** (1909). Toxicity: R. I. Mrongovius *et al., Clin. Exp. Pharmacol. Physiol.* **3**, 443 (1976).

Practically tasteless needles, mp 147-149°. Sublimes. Slightly sol in cold water, but freely sol in hot water. Readily sol in alcohol, ether and in alkaline solns. LD_{50} in male mice (mmoles/kg): 3.25 i.p. (Mrongovius).

Caution: This substance may be habit forming and is listed in the U.S. Code of Federal Regulations, Title 21 Part 329.1 (1995).

THERAP CAT: Sedative, hypnotic.

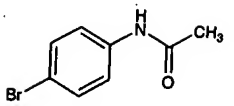
1419. *N*-Bromoacetamide. Acetobromamide. C_2H_4BrNO: mol wt 137.96. C 17.41%, H 2.92%, Br 57.92%, N 10.15%, O 11.60%. $CH_3CONHBr$. Prepd by treating a cooled (0-5°) soln of acetamide dissolved in bromine with ice cold aq 50% KOH, allowing to stand for 2-3 hr at 0-5°, saturating with NaCl, and extracting with chloroform: Oliveto, Gerold, *Org Syn.* **31**, 17 (1951).

Needles from chloroform + hexane, mp 102-105°. Sol in warm water, freely sol in cold ether. Unstable to light and heat.

Monohydrate, rectangular plates from hydr ether, mp 70-75°. Freely sol in cold water, alcohol, ether, less freely in chloroform.

USE: Brominating agent; in oxidation of primary and secondary alcohols.

1420. *p*-Bromoacetanilide. *N-(4-Bromophenyl)acetamide; 4'-bromoacetanilide;* monobromoacetanilide; bromo-Antisepsin; Asepsin; Bromoantifebrin. C_8H_8BrNO; mol wt 214.06. C 44.89%, H 3.77%, Br 37.33%, N 6.54%, O 7.48%. Prepd by bromination of acetanilide: Remmers, *Ann.* **7**, 346 (1874); Merker, Vona, *J. Chem. Ed.* **26**, 613 (1949); Kn[illegible] Monsanto).



Crystals, from 95% alc, mp 168° (with previous softening). d 1.72. Practically insol in cold water. Sparingly sol in hot water; sol in benzene, chloroform, ethyl acetate; moderately sol in alcohol.

THERAP CAT: Analgesic, antipyretic.

1421. Bromoacetic Acid. $C_2H_3BrO_2$; mol wt 138.95. C 17.29%, H 2.18%, Br 57.51%, O 23.03%. $BrCH_2COOH$. Prepn by bromination of acetic acid: Perkin, Duppa, *Ann.* **108**, 106 (1858); from chloroacetic acid and HBr: Lake, Asadorian; Asadorian, Burk, U.S. pats. **2,553,518; 3,130,222** (1951, 1964, both to Dow); from glycolic acid and HBr: Johnston, U.S. pat. **2,876,255** (1959 to Ethyl Corp.).

Hygroscopic crystals, mp 50°. bp 208°. d 1.93. Very sol in water, alcohol. *Protect from air and moisture.*

Caution: Irritant and corrosive to skin, mucous membranes.

1422. Bromoacetone. *1-Bromo-2-propanone.* C_3H_5BrO; mol wt 136.98. C 26.31%, H 3.68%, Br 58.33%, O 11.68%. CH_3COCH_2Br. Prepn by bromination of acetone: Emmerling, Wagner, *Ann.* **204**, 29 (1880); Catch *et al., J. Chem. Soc.* **1948**, 272; Ross U.S. pat. **2,452,154** (1948 to Colgate-Palmolive-Peet); by bromination acetone enol acetate: Magerlein, U.S. pat. **2,752,341** (1956 to Upjohn).

Liquid, mp −36.5°. bp 137°, bp_{50} 63.5-64°. d^{23} 1.634. n_D^{15} 1.4697. Turns violet rapidly even in absence of air. Sparingly sol in water; sol in alcohol, acetone. *Keep tightly closed and protected from light.*

Caution: Violent lacrimator.

USE: Chemical war gas.

1423. *p*-Bromoacetophenone. *1-(4-Bromophenyl)ethanone;* methyl *p*-bromophenyl ketone. C_8H_7BrO; mol wt 199.05. C 48.27%, H 3.54%, Br 40.14%, O 8.04%. $BrC_6H_4COCH_3$. Prepd from bromobenzene and acetic anhydride in CS_2 in the presence of anhydr $AlCl_3$: Adams, Noller, *Org. Syn.* **5**, 17 (1925).

Leaflets from alc. mp 54°. bp_{736} 255.5°; bp_{15} 130°; bp_7 117°. Easily volatile with steam. Sol in alcohol, ether, glacial acetic acid, benzene, petr ether, carbon disulfide.

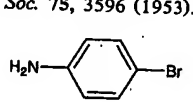
Oxime, C_8H_8BrNO, needles from dil alc, mp 128.5°.

1424. ω-Bromoacetophenone. *2-Bromo-1-phenylethanone;* phenacyl bromide. C_8H_7BrO; mol wt 199.05. C 48.27%, H 3.54%, Br 40.14%, O 8.04%. $C_6H_5COCH_2Br$. Prepn from acetophenone and bromine: Rother, Reid, *J. Am. Chem. Soc.* **41**, 77 (1919); Cowper, Davidson, *Org. Syn.* **19**, 24 (1939); Shevchuk, Dombrovskii, *Zh. Obshch. Khim.* **33**(4), 1135 (1963).

Crystals, mp 50°. d 1.65. bp_{20} 133-135°. Irritating vapors. *Lacrimator!* Practically insol in water. Freely sol in alcohol, benzene, chloroform, ether.

Caution: Highly irritating to skin, eyes, mucous membranes.

1425. *p*-Bromoaniline. *4-Bromobenzeneamine;* 4-bromoaniline. C_6H_6BrN; mol wt 172.02. C 41.89%, H 3.52%, Br 46.45%, N 8.14%. Prepd by steam distilling sodium hydroxide and *p*-bromoacetanilide: Scott, *J. Chem. Soc.* **123**, 3199 (1923); by direct bromination of aniline: Kosolapoff, *J. Am. Chem. Soc.* **75**, 3596 (1953).



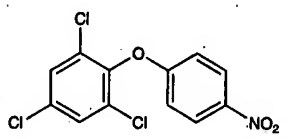
Rhombic crystals from dil alc; mp 66-66.5°. $d_4^{99.6}$ 1.4970 (liq). Very sol in alcohol and ether. Insol in cold water. pKb (25°): 10.28 also reported as 9.98.

USE: In prepn of azo dyes; condensed with formaldehyde in prepn of dihydroquinazolines.

Fifth Annual Report on Carcinogens (NTP 89-239, 1989) p 340.

THERAP CAT: Antineoplastic.

2158. Chlornitrofen. *1,3,5-Trichloro-2-(4-nitrophenoxy)benzene;* 4-nitrophenyl-2,4,6-trichlorophenyl ether; 2,4,6-trichlorophenyl-4′-nitrophenyl ether; CNP; MO-338; MO; Showrone. $C_{12}H_6Cl_3NO_3$; mol wt 318.54. C 45.25%, H 1.90%, Cl 33.39%, N 4.40%, O 15.07%. Prepd, not claimed: Neth. pat. Appl. 6,405,727; I. Takayuki *et al.*, U.S. pat. 3,316,080 (1964, 1967 both to Mitsui Chemical); and field studies: G. W. Mason, E. G. Brooker, *Proc. 21st N. Z. Weed Pest Control Conf.* 163 (1968). Properties: J. Kanazawa, *Pestic. Sci.* 12, 417 (1981). Absorption and metabolism in crops: H. Shimotori, S. Kuwatsuka, *J. Pestic. Sci.* 3, 267 (1978). Degradation studies: M. Oyamada, S. Kuwatsuka, *ibid.* 4, 157 (1979). GC determn in fish: T. Yamagishi *et al., Bull. Environ. Contam. Toxicol.* 23, 57 (1979); in river water: H. Kobayashi *et al., J. Chromatog.* 643, 197 (1993).



Pale yellow needles from ethanol, mp 105-106°. Soly: 0.25 mg/l water. Soly at 20-25°: 764 μg/l water. Vapor pressure (109°): 4.67×10^4 mPa. Partition coefficient (octanol/water): 4709. LC_{50} in carp (48 hr): >10,000 μg/l; LC_{50} in flea (3 hr): >40,000 μg/l (Kanazawa).

USE: Herbicide.

2159. Chloroacetaldehyde. 2-Chloro-1-ethanal; monochloroacetaldehyde. C_2H_3ClO; mol wt 78.50. C 30.60%, H 3.85%, Cl 45.16%, O 20.38%. $ClCH_2CHO$. Prepd industrially by carefully controlled chlorination of acetaldehyde: Söll, Ger. pat. 844,595 (1943 to Knapsack-Griesheim); Shawinigan Chem. Ltd., Brit. pat. 644,914 (1947); Guinot, Tabuteau, *Compt. Rend.* 231, 234 (1950); Fr. pat. 1,012,991 (1950 to Comp. des Prod. Chim. et Electrometallurg.).

Liquid. Acrid, penetrating odor. bp_{760} 85-86°. The anhydr substance polymerizes on standing but reverts to the monomer on distillation.

Hemihydrate, probably $ClCH_2CH(OH)OCH(OH)CH_2Cl$, platelets from water, mp 43-50°, bp 85.5° with decompn into water and chloraldehyde. Sol in water, alcohol, ether.

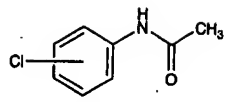
Caution: Potential symptoms of overexposure are irritation of eyes, skin and mucous membranes; skin burns; eye damage; pulmonary edema; skin, respiratory system sensitization; narcosis, coma. *See NIOSH Pocket Guide to Chemical Hazards* (DHHS/NIOSH 90-117, 1990) p 64.

USE: In the manufacture of 2-aminothiazole; to facilitate bark removal from tree trunks.

2160. Chloroacetamide. *2-Chloroacetamide.* C_2H_4ClNO; mol wt 93.51. C 25.69%, H 4.31%, Cl 37.91%, N 14.98%, O 17.11%. $ClCH_2CONH_2$. Prepn from ethyl chloroacetate and ammonia: Jacobs, Heidelberger, *Org. Syn.* coll. vol. I, 153 (2nd ed., 1941); from chloroacetyl chloride and ammonium acetate: Finan, Fothergill, *J. Chem. Soc.* 1962, 2824.

Crystals from water, mp 119-120°. bp ~225° (dec). Sol in 10 parts water, 10 parts abs alc; very slightly sol in ether. Two isomorphous crystalline modifications: α-form, "stable form", obtained by sublimation and by crystallization from nonpolar solvents; β-form, "unstable form", obtained by quenching the melt or by crystallization from polar solvents, *see* Katayama, *Acta Crystallogr.* 9, 986 (1956); 10, 468 (1957); B. Kalyanaraman *et al., J. Cryst. Mol. Struct.* 8, 175 (1978).

2161. Chloroacetanilide. C_8H_8ClNO; mol wt 169.61. C 56.65%, H 4.75%, Cl 20.90%, N 8.26%, O 9.43%. Prepn of *m-*, *o-* and *p*-isomers: Roberts *et al., J. Org. Chem.* 24, 654 (1959). Sepn of *o-* and *p*-isomers: Orton, Bradford, *J. Chem. Soc.* 1927, 986.



m-Chloroacetanilide, N-(3-chlorophenyl)acetamide; chloroacetanilide. Needles from 50% glacial acetic acid, mp 77-78°. Readily sol in alcohol, benzene, carbon disulfide; very slightly sol in ligroin. uv max (95% ethanol): 245 nm (log ε 4.19).

o-Chloroacetanilide. Needles from dil glacial acetic acid, mp 87-88°. Sublimes at about 50-60°. Practically insol in water, alkalies; sol in alc; more sol in benzene than corresponding *p*-isomer. uv max (95% ethanol): 240 nm (log ε 4.02).

p-Chloroacetanilide. Orthorhombic crystals from aq acetic acid, alc, or acetone. mp 178-179°. d_4^{25} [illegible]. Practically insol in water; readily sol in alc, ether, carbon disulfide; slightly sol in CCl_4, benzene. uv max (95% ethanol): 249 nm (log ε 4.25).

2162. Chloroacetic Acid. Chloroethanoic acid; monochloroacetic acid; MCA. $C_2H_3ClO_2$; mol wt 94.50. C 25.42%, H 3.20%, Cl 37.52%, O 33.86%. $ClCH_2COOH$. Made by chlorination of glacial acetic acid in presence of small amount of sulfur or iodine; also by hydrolysis of chlorethylene with 90% H_2SO_4. Lab prepn: Gattermann-Wieland, *Praxis des organischen Chemikers* (de Gruyter, Berlin, 40th ed., 1961) pp 109, 110. Manuf: Faith, Keyes, Clark's *Industrial Chemicals*, F. A. Lowenheim, M. K. Moran, Eds. (Wiley-Interscience, New York, 4th ed., 1975) pp 254-257. Toxicity: Woodward *et al., J. Ind. Hyg. Toxicol.* 23, 78 (1941).

Colorless or white, deliquesc crystals. d 1.580. Exists in α, β and γ forms having mp 63°, 55-56° and 50° respectively; mp for acid of commerce is 61-63°. bp for all forms is 189°. Very sol in water; sol in alcohol, benzene, chloroform, ether. *Keep well closed and in a cool place.*

Sodium salt, $C_2H_2ClNaO_2$, *Monoxone.* White crystalline solid. Soly in water at 20°: 85 g/100 ml. LD_{50} in mice, guinea pigs (mg/kg): 76, 255, 80 orally (Woodward).

Caution: Irritating to skin, mucous membranes.

USE: Herbicide. Manuf various dyes and other organic chemicals.

2163. Chloroacetic Anhydride. *Chloroacetic acid anhydride;* monochloroacetic acid anhydride; *sym*-dichloroacetic anhydride. $C_4H_4Cl_2O_3$; mol wt 170.98. C 28.10%, H 2.36%, Cl 41.47%, O 28.07%. $(ClCH_2CO)_2O$. Prepd by treating monochloroacetic acid in tetrahydrofuran with methoxyacetylene at 15° and distilling the mixture: Ralston *et al., J. Chem. Soc.* 1954, 1860; by mixing monochloroacetic acid and HCN with 1,4-dioxane satd with HCl and fractionating the mixture: Krieble, Smellie, U.S. pat. 390,106 (1945); by heating chloroacetyl chloride and monochloroacetic acid in the presence of $AlCl_3$: Strossa, Schwegler, U.S. pat. 1,713,104 (1929 to Dow).

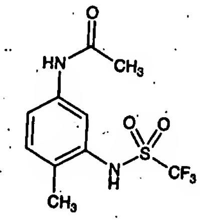
Prisms from benzene; d_4^{20} 1.5494, mp 46°. bp_{760} 203°; 163°; bp_{62} 149°; bp_{24} 126°; bp_{10} 109-110°; $bp_{0.05}$ 118-12[illegible]. Freely sol in ether, chloroform; slightly sol in benzene; practically insol in cold ligroin.

USE: In *N*-acetylation of amino acids in alkaline soln; prepn of cellulose chloroacetates.

2164. Chloroacetone. *1-Chloro-2-propanone;* chlorketone; monochloroacetone; monochloracetone; acetonyl chloride; chloropropanone; 1-chloro-2-ketopropane; 1-chloro-2-oxopropane. C_3H_5ClO; mol wt 92.52. C 38.94%, H 5.[illegible], Cl 38.32%, O 17.29%. $ClCH_2COCH_3$. Prepn by the action of chlorine upon diketene: A. B. Boese, Jr., U.S. pat. 2,209,683 (1940 to Carbide and Carbon Chem.); by chlorination of acetone: E. J. Rahrs, U.S. pat. 2,235,562 (1941 to Eastman Kodak); G. H. Morey, U.S. pat. 2,240,[illegible] (1941 to Commercial Solvents Corp.). Stabilization: [illegible], U.S. pat. 2,229,651 (1941 to Commercial Solvents); E. J. Rahrs, U.S. pat. 2,263,010 (1941 to Eastman Kodak). Forms binary azeotropes with many organic liquids: [illegible] Horsley, *Azeotropic Data,* Advances in Chemistry Series 6 (Washington, 1952) p 73. Reacts with aryl [illegible]

Fluora

40.54%, H 3.74%, F 19.24%, N 9.46%, O 16.20%, S 10.82%. Prepn: Harrington *et al.*, U.S. pat. 3,639,474 (1972 to Minnesota Mining & Mfg).



mp 175-176.5°.

USE: Plant growth retardant.

4199. Fluorine. F; at. wt 18.9984032; at. no. 9; valence 1; elemental state F_2. A halogen. Occurrence in earth's crust 0.065% by wt. Natural abundance of isotopes: ^{19}F 100%; ^{18}F ($T_{1/2}$ 109.7 min) is prepared in nuclear reactors. Does not occur in elemental state in nature. Most important sources are fluorite, cryolite, and fluorapatite: Finger, "Fluorine Resources and Fluorine Utilization" in *Advances in Fluorine Chemistry*, vol. 2, M. Stacey *et al.*, Eds. (Butterworths, London, 1961) pp 35-54. Discovered in 1771 by Scheele. Isolated in 1886 by electrolyzing a soln of potassium fluoride in anhydr hydrogen fluoride at −23°, using platinum-iridium electrodes: Moissan, *Compt. Rend.* **102,** 1543 (1886); **103,** 202, 256. Subsequent methods of prepn: Ruff, *Ber.* **69,** 181 (1936); Henne, *J. Am. Chem. Soc.* **60,** 96 (1938). ^{18}F has been used to study fluorine exchange reactions: Dave, Sowerby, "Isotopic Halogen Exchange Reactions" in *Halogen Chemistry*, vol. 1, V. Gutmann, Ed. (Academic Press, New York, 1967) pp 41-132. Toxicity data: Keplinger, Suissa, *Am. Ind. Hyg. Assoc. J.* **29,** 10 (1968). *Reviews:* A. J. Rudge, *The Manufacture and Use of Fluorine and its Compounds* (Oxford University Press, 1962); O'Donnell, "Fluorine" in *Comprehensive Inorganic Chemistry*, vol. 2, J. C. Bailar, Jr. *et al.*, Eds. (Pergamon Press, Oxford, 1973) pp 1009-1106; A. J. Woytek in Kirk-Othmer *Encyclopedia of Chemical Technology* vol. 10 (Wiley-Interscience, New York, 3rd ed., 1980) pp 630-654.

Pale yellow, diatomic gas. mp −219.61° (53.54°K); bp −188.13° (85.02°K); d (liq, −188.13°) 1.5127; vapor pressure data: Hu *et al.*, *J. Am. Chem. Soc.* **75,** 5642 (1953); White *et al.*, *ibid.* **76,** 2584 (1954). Crit temp: −129°; crit pressure: 55 atm. Most reactive nonmetal; higher oxidation potential than ozone; most electronegative element; E° (calc) ½F/F⁻ 2.9 V. F-F bond weaker than Cl-Cl and Br-Br bonds; enthalpy of dissociation: 37.7 kcal. Reacts vigorously with most oxidizable substances at room temp, frequently with ignition. Combines directly or indirectly, to form fluorides with all the elements except helium, neon and argon. Dec water, giving hydrofluoric acid, HF, oxygen fluoride, OF_2, hydrogen peroxide, oxygen and ozone. Reacts with nitric acid, forming the explosive gas, fluorine nitrate, NO_3F; with sulfuric acid, giving fluorosulfuric acid, $HFSO_3$. Yields the metal fluorides, water, oxygen and oxygen fluoride when made to react with metal hydroxides in the cold. Reacts violently with organic compds, usually with disintegration of the molecule. Under controlled conditions, however, hydrocarbon vapors may be fluorinated with elemental fluorine. Solid fluorine explodes when brought in contact with liquid hydrogen. Under ordinary conditions it does not react directly with oxygen, nor does it react with oxides of sodium, potassium or calcium. LC_{50} (1 hr) inhalation by rats, mice, guinea pigs: 185, 150, 170 ppm (by vol) (Keplinger, Suissa).

Caution: Potential symptoms of overexposure are irritation of eyes, nose and respiratory tract; laryngeal spasms, bronchial spasms; pulmonary edema; eye and skin burns. *See NIOSH Pocket Guide to Chemical Hazards* (DHHS/NIOSH 90-117, 1990) p 116. Chronic ingestion of high concentrations from water supply can cause mottled enamel of teeth and osteosclerosis. *See Clinical Toxicology of Commercial Products*, R. E. Gosselin *et al.*, Eds. (Williams & Wilkins, Baltimore, 5th ed., 1984) Section III, pp 185-193.

4200. Fluorine Dioxide. Dioxygen difluoride. mol wt 70.00. F 54.28%, O 45.72%. Prepd from O... Ruff, Menzel, *Z. Anorg. Allgem. Chem.* **211,** ... **217,** 85 (1934); Goetschel *et al.*, *J. Am. Chem. Soc.* ... (1969). Chemical behavior: Streng, *ibid.* **85,** ... *Review:* Kemmitt, Sharp, *Advan. Fluorine Chem.* ... (1965).

Thermally unstable gas at room temp; begins to ... fluorine and oxygen at −100°. Pale yellow solid ... low liq at low temp. mp −154°C (119°K). Early ... described as brown gas, red liq and orange ... −163.5°), probably contained other fluorine-oxygen ... pounds as impurities: Goetschel *et al.*, *loc. cit.* ... dizing and fluorinating agent.

4201. Fluorine Monoxide. Oxygen fluoride; ... oxide. F_2O; mol wt 54.00. F 70.37%, O 29.63%. ... passing fluorine slowly through an aq NaOH soln ... *Inorg. Syn.* **1,** 109 (1939); Schnizlein *et al.*, *J. Phys. Chem.* **56,** 233 (1952). Toxicity data: Darmer *et al.*, *Am. ... Assoc. J.* **33,** 661 (1972). *Review:* Kemmitt, Sharp, ... *Fluorine Chem.* **4,** 213-314 (1965).

Colorless gas. Yellowish-brown when liq. Peculiar ... Does not attack glass in the cold. Corrodes ... Reacts very slowly with water. The gas may be ... water unchanged for a month. d (liq; −224°) 1.90 ... −223.8°. bp −145.3°. Trouton constant 20.65. ... water (0°) 6.8 ml gas/100 ml H_2O. LC_{50} (1 hr) inhalation ... rats, mice: 2.6, 1.5 ppm (Darmer).

Caution: Potential symptoms of overexposure are ... table headache; respiratory system irritation, pulmonary edema; eye and skin burns. *See NIOSH Pocket Guide to Chemical Hazards* (DHHS/NIOSH 90-117, 1990) p 17...

4202. Fluorine Nitrate. *Nitroxy fluoride;* ... oxyfluoride; nitryl hypofluorite. FNO_3; mol wt 81.00 ... 23.45%, N 17.29%, O 59.25%. $FONO_2$. Prepd by the ... tion of fluorine on nitric acid: Cady, *J. Am. Chem. Soc.* ... 2635 (1934); Ruff, Kwasnik, *Angew. Chem.* **48,** 238 (19..); Kwasnik in *Handbook of Preparative Inorganic Chemistry* vol. 1, G. Brauer, Ed. (Academic Press, New York, ... 1963) pp 187-189. *Reviews:* Kemmitt, Sharp, *Advan. Fluorine Chem.* **4,** 216-218 (1965); Woolf, *ibid.* **5,** 1-30 (19..); Schmutzler, *Angew. Chem. Int. Ed.* **7,** 440-455 (1968).

Colorless gas. Moldy, acrid odor. mp −175° ... −45.9°. d (liq at bp) 1.507. $d^{-193.2}$ (solid) 1.951. Trouton const 20.8. The liq explodes on slight percussion. Hydrolyzed by water to OF_2, O_2, HF and HNO_3. Sol in acetone. Conflagrates on contact with alcohol, ether, aniline. May be stored in vacuum-sealed glass ampuls cooled by liq oxygen. Powerful oxidizing agent.

USE: Oxidizing agent in rocket propellants.

4203. Fluorine Perchlorate. Chlorine tetroxyfluoride. $ClFO_4$; mol wt 118.45. Cl 29.93%, F 16.04%, O 54.0...% $FOClO_3$. Prepd by passing fluorine over cold 72% aq perchloric acid in platinum apparatus: Rohrback, Cady, *J. Am. Chem. Soc.* **69,** 677 (1947).

Colorless gas. Pungent, acrid odor. mp −167.3°, bp −15.9°. Explodes on the slightest provocation, *i.e.*, on contact with rough surfaces, dust, grease, rubber, on melting, distilling, etc.

Caution: Attacks lungs even in traces.

4204. Fluoroacetamide. Fluoroacetic acid amide; monofluoroacetamide; 1081; Fluorakil 100; Fussol. C_2H_4FNO; mol wt 77.06. C 31.17%, H 5.23%, F 24.65%, N 18.18%, O 20.76%. CH_2FCONH_2. Numerous syntheses, e.g. from fluoroacetyl chloride and NH_3: Truce, *J. Am. Chem. Soc.* **70,** 2828 (1948). Mode of action study and toxicity data: F. Matsumura, R. D. O'Brien, *Biochem. Pharmacol.* **12,** 1201 (1963).

Crystals. Sublimes on heating. Freely sol in water, sol in acetone; sparingly sol in chloroform. LD_{50} i.p. in mice: ... mg/kg (Matsumura, O'Brien).

USE: Rodenticide. Insecticide proposed mainly for use on fruits to combat scale insects, aphids, and mites.

4205. Fluoroacetic Acid. Fluoroethanoic acid; gifblaar poison. $C_2H_3FO_2$; mol wt 78.04. C 30.78%, H 3.87%, F 24.34%, O 41.00%. CH_2FCOOH. Occurs in "Gifblaar" ...

Consult the Name Index before using this section.

X

Dichapetalum cymosum (Hook.) Engl. (*Chailletia cymosa* Hook.), *Dichapetalaceae*, one of the most poisonous plants, habitat South Africa. Prepd from methyl iodoacetate by heating with silver fluoride or mercurous fluoride or from methyl chloroacetate by heating with potassium fluoride, followed by saponification of the methyl ester with baryta: Barris, *Bull. Soc. Chim.* [3] **15**, 1134 (1896); Gryszkiewicz-Trochimowski *et al.*, *Rec. Trav. Chim.* **66**, 419 (1947). Review and biochemical aspects: Peters, *Endeavour* **13**, 147 (1954); Peters *et al.*, *Biochem. J.* **77**, 17 (1960). Reviews of toxicity: *Clinical Toxicology of Commercial Products*, R. E. Gosselin *et al.*, Eds. (Williams & Wilkins, Baltimore, 4th ed., 1976) Section III, pp 163-166; Gribble, *J. Chem. Ed.* **50**, 460-462 (1973).

Crystals. Burns with a green flame.

Sodium salt, $C_2H_2FNaO_2$, *Compd 1080, 1080, Fratol.* A white powder, sol in water. Oral lethal dose estimated to be 2-5 mg/kg body wt.

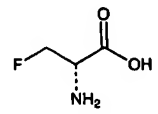
Methyl ester, $CH_2FCOOCH_3$, liq, odor of ethyl acetate. n_D 1.1613. bp 104.5°. Sol in water; slightly sol in petr ether.

Ethyl ester, $CH_2FCOOC_2H_5$, liq, odor of ethyl acetate. d_4^{20} 1.0926. bp_{758} 121.6°. $n_D^{20.5}$ 1.3767. Sol in water.

Caution: Extremely toxic. Causes convulsions and ventricular fibrillation. Should never be used where inhalation or food contamination may occur. Locally, it is an irritant.

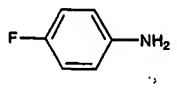
USE: The sodium salt is used as a water-soluble rodenticide.

4206. 3-Fluoro-D-alanine. *S*-2-Amino-3-fluoropropanoic acid; FA. $C_3H_6FNO_2$; mol wt 107.08. C 33.65%, H 5.65%, F 17.74%, N 13.08%, O 29.88%. Synthesis by photofluorination of D-alanine or by resolution of the DL-form: J. Kollonitsch, F. M. Kahan; J. Kollonitsch, Ger. pat. 2,136,067; 2,229,245, *C.A.* **76**, 100053p (1972); **78**, 72386d (1973); *idem*, U.S. pat. 3,839,170 (1972, 1972, 1974 all to Merck & Co.). The 2-deuterated analog has also been prepared. Antibacterial activity: J. Kollonitsch *et al.*, *Nature* **243**, 346 (1973).



Crystals from water, dec 168°. $[\alpha]_D^{20}$ −10° (c = 3 in 1*N* HCl). Stable at acid pH; less stable at pH > 8.

4207. *p*-Fluoroaniline. *4-Fluorobenzenamine.* C_6H_6FN; mol wt 111.12. C 64.85%, H 5.44%, F 17.10%, N 12.61%. Prepd by reduction of 1-fluoro-4-nitrobenzene by Raney nickel: Benington *et al.*, *J. Org. Chem.* **18**, 1508 (1953); Finger *et al.*, *J. Am. Chem. Soc.* **81**, 98 (1959); by saturated sodium borohydride: Lalancette, Brindle, *Can. J. Chem.* **49**, 2990 (1971).



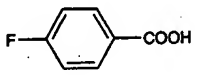
Liquid. d_4^{20} 1.1725; d_4^{25} 1.1690. mp −1.9°. bp 188°, bp_{20} 71°. n_D 1.51954. Heat of combustion 780.4 kcal/mol. Slightly sol in water.

USE: Intermediate in the manuf of herbicides and plant growth regulators.

4208. Fluorobenzene. C_6H_5F; mol wt 96.10. C 74.97%, H 5.24%, F 19.77%. Obtained by warming benzenediazonium chloride with concd HF. Solubility value: Dreisbach *et al.*, *J. Am. Chem. Soc.* **55**, 650 (1933). Pressure, vapor temp constants: Douslin *et al.*, *J. Am. Chem. Soc.* **80**, 2001 (1958).

Liquid, benzene odor. d_4^{20} 1.024. bp_{760} 84.73°; $bp_{13\ atm}$ 275°. mp −40°. n_D^{20} 1.4677. Miscible with ether. Sol (30°C): 1.54 g/1000 g water.

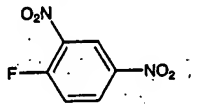
4209. *p*-Fluorobenzoic Acid. $C_7H_5FO_2$; mol wt 140.11. C 59.99%, H 3.60%, F 13.56%, O 22.84%. Prepd by treating *p*-carbethoxybenzenediazonium chloride with fluoboric acid followed by thermal decompn of the resulting *p*-carbethoxybenzenediazonium fluoborate and by hydrolysis of the ensuing ethyl ester of *p*-fluorobenzoic acid: Schiemann, Winkelmüller, *Org. Syn.* **13**, 52 (1933).



Monoclinic prisms from water. Peculiar sweet taste. mp 182.6°. Sparingly sol in cold water; freely sol in hot water. Soly in water at 32°: 1.1 g/l. Water solns evaporate without leaving a residue. Sol in alc, ether. pK (25°): 3.85.

Ethyl ester, $C_9H_9FO_2$, crystals, mp 26°. bp 210°.

4210. 1-Fluoro-2,4-dinitrobenzene. 2,4-Dinitro-1-fluorobenzene; DNFB; FDNB; Sanger's reagent. $C_6H_3FN_2O_4$; mol wt 186.10. C 38.72%, H 1.62%, F 10.21%, N 15.05%, O 34.39%. Prepn: A. F. Holleman, J. W. Beekman, *Rec. Trav. Chim.* **23**, 225 (1904); Cook, Saunders, *Biochem. J.* **41**, 558 (1947). Use in peptide analysis: Sanger, *Biochem. J.* **39**, 507 (1945); **40**, 261 (1946); **45**, 563 (1949); Porter, Sanger, *ibid.* **42**, 287 (1948). Tumor promoting activity: F. G. Bock *et al.*, *Cancer Res.* **29**, 179 (1969). Mutagenicity study: D. R. Jagannath *et al.*, *Mutat. Res.* **78**, 91 (1980). Review of uses: *Reagents for Organic Synthesis*, L. F. Fieser, M. Fieser, Eds. (Wiley, New York, 1967) pp 321-322.



Pale yellow crystals from ether, mp 26°. $bp_{2.0}$ 137°. Sol in benzene, ether, propylene glycol.

Caution: Vesicant. For proper handling see: J. S. Thompson, O. P. Edmunds, *Ann. Occup. Hyg.* **23**, 27 (1980).

USE: Reagent for labeling a terminal amino acid group; in modified Wohl degradations of aldoses. As hapten.

4211. Fluoroform. *Trifluoromethane.* CHF_3; mol wt 70.01. C 17.16%, H 1.44%, F 81.41%. Prepd from $CHCl_3$ and HF: Meslans, *Compt. Rend.* **110**, 717 (1890); Valentiner, U.S. pat. **643,835** (1900); Ruff, *Ber.* **69**, 299 (1936); Whallay, *J. Soc. Chem. Ind. (London)* **66**, 429 (1947); Kwasnik in *Handbook of Preparative Inorganic Chemistry* vol. 1, G. Brauer, Ed. (Academic Press, New York, 2nd ed., 1963) pp 204-205. Early industrial prepn: Pearlson in *Fluorine Chemistry* vol. I, J. H. Simons, Ed. (Academic Press, New York, 1950) p 467.

Colorless, odorless gas. Stable up to 1150°. Chemically very inert. d (solid) 1.935. d (liq; −100°) 1.52. mp −160°. bp −84.4°. Critical temp 33°, critical pressure 47 atm, critical density 0.516. May be stored over water. Practically non-toxic: N. V. Sidgwick, *The Chemical Elements and Their Compounds*, vol. II (Oxford, 1950) p 1130.

Caution: May be slightly irritating to respiratory tract, and, in high concns, narcotic.

USE: Refrigerant for low temps.

4212. Fluoromethane. Methyl fluoride. CH_3F; mol wt 34.03. C 35.29%, H 8.88%, F 55.82%. Prepn in 82% yield by heating fluorosulfonic acid methyl ester with KF: Zappel, Jonas, Ger. **1,131,197** (1962 to Bayer).

Gas. Agreeable ether-like odor. Burns with evolution of HF, the flame being about as colorless as that of alc: Dumas, Peligot, *Ann.* **15**, 59 (1835). d (liq; −78°) 0.8774. d (gas) 1.1951 (air = 1); d (gas) 1.0813 (oxygen = 1). Dipole moment 1.81. Molecular volume: 22.03. Van der Waals constants: 0.00923; 0.002350. Critical temp 44.9°; crit press. 62.0 atm. Dielectric constant (for wavelengths below 10^4 cm) = 1.00948. mp −141.8°. bp_{672} −75.7°; bp_{760} −78.2°. bp_{143} −103.7°. One hundred vols of water dissolve 166 vols of the gas at 15°. Freely sol in alcohol, ether.

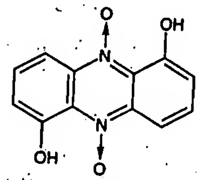
Caution: Narcotic in high concns.

4213. Fluorometholone. *(6α,11β)-9-Fluoro-11,17-dihydroxy-6-methylpregna-1,4-diene-3,20-dione;* 21-desoxy-9α-fluoro-6α-methylprednisolone; 21-desoxy-6α-methyl-9α-fluoroprednisolone; fluormetholon; Cortilet; Delmeson; Effludimex; Fluaton; Flumetholon; FML; Loticort; Oxylone;

iodo aceti

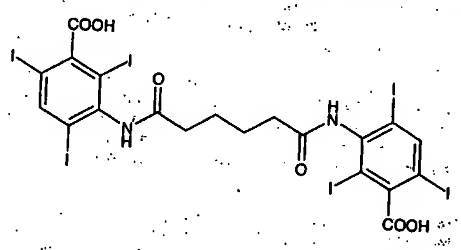
USE: Chlorinating and oxidizing agent.
THERAP CAT: Anti-infective (topical).

5042. Iodinin. *1,6-Phenazinediol 5,10-dioxide;* 1,5-dihydroxyphenazine *N,N'*-dioxide. $C_{12}H_8N_2O_4$; mol wt 244.21. C 59.02%, H 3.30%, N 11.47%, O 26.21%. Antibiotic pigment from *Chromobacterium iodinum:* Clemo, McIlwain, *J. Chem. Soc.* **1938**, 479; Hegedüs in *Emil Barell Jubilee Volume* (1946), p 388; from *Waksmania aerata* and *Pseudomonas iodinum:* Gerber, Lechevalier, *Biochemistry* **3**, 598 (1964). Structure: Clemo, Daglish, *J. Chem. Soc.* **1950**, 1481. Synthesis: Matsumura, Takeda, *Nippon Kagaku Zasshi* **81**, 515 (1960), *C.A.* **56**, 470a (1962). Biosynthetic studies on incorporation of shikimic acid, *q.v.*, into iodinin: U. Hollstein *et al.*, *Tetrahedron Letters* **1978**, 2987; R. B. Herbert *et al.*, *J. Chem. Soc. Perkin Trans. I* **1979**, 2411; T. Etherington *et al.*, *ibid.* 2416.



Purple crystals with coppery sheen from chloroform, dec 236°. pK 12.5. Stable in acid, unstable in alkali. Sol in benzene, toluene, xylene, carbon disulfide, chloroform, ethyl acetate. Slightly sol in hot alc. Practically insol in cold alc, ether, acetic acid, petr ether, amyl alcohol. Insol in water. Sol in concd sulfuric acid and in glacial acetic acid with red color. Sol in NaOH solns giving brilliantly blue solns which deposit green crystals of the unstable sodium deriv.

5043. Iodipamide. *3,3'-[(1,6-Dioxo-1,6-hexanediyl)diimino]bis[2,4,6-triiodobenzoic acid]; 3,3'-(adipoyldiimino)bis[2,4,6-triiodobenzoic acid]; N,N'*-adipylbis(3-amino-2,4,6-triiodobenzoic acid); adipic acid di(3-carboxy-2,4,6-triiodoanilide); adipiodone; Cholografin; Cholospect. $C_{20}H_{14}I_6N_2O_6$; mol wt 1139.77. C 21.08%, H 1.24%, I 66.81%, N 2.46%, O 8.42%. Prepn: Priewe, Rutkowski, U.S. pat. **2,776,241** (1957 to Schering AG); Kotler-Brajtburg *et al.*, *Roczniki Chem.* **36**, 763 (1962), *C.A.* **58**, 5568a (1963). Purification: Cassebaum, Drux, East Ger. pat. **33,738** (1964), *C.A.* **63**, 11441b (1965). Pharmacology: Fischer, Varga, *Acta Physiol.* **38**, 135 (1970). Metabolic studies: Kiyono *et al.*, *Radioisotopes* **20**, 78 (1971). Pharmacology and toxicity study: F. J. Rosenberg *et al.*, *Invest. Radiol.* **15**, S142 (1980). Comprehensive description: H. H. Lerner in *Analytical Profiles of Drug Substances* vol. 3, K. Florey, Ed. (Academic Press, New York, 1974) pp 333-363.



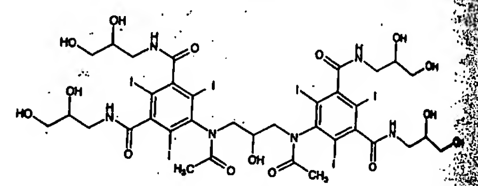
Crystals, dec 306-308°. $n_D^{21.5}$ 1.3294 (c = 0.445 in methanol). Soly at 20°: methanol 0.8%; ethanol 0.3%; acetone 0.2%; ether 0.1%. Practically insol in water, benzene. LD_{50} in mice, rats (mg/kg): 2380 ±290, 4430 ±310 i.v. (Rosenberg).

Disodium salt, $C_{20}H_{12}I_6N_2Na_2O_6$. Prepd by dissolving the free acid in a dil aq soln of NaOH and buffering to pH 6.5-7.7. Used as 20% soln.

Bis[*N*-methylglucamine] salt, $C_{34}H_{48}I_6N_4O_{16}$, *iodipamide meglumine, Biligrafin, Cholegrafin, Endocistobil, Endografin, Intrabilix, Transbilix.* More sol in water than the disodium salt. Used as 40% soln.

cystographic).
THERAP CAT (VET): Diagnostic aid (radiopaque medium).

5044. Iodixanol. *5,5'-[(2-Hydroxy-1,3-propanediyl)bis(acetylimino)]bis[N,N'-bis(2,3-dihydroxypropyl)-2,4,6-triiodo-1,3-benzenedicarboxamide];* 5,5'-[(2-hydroxytrimethylene)bis(acetylimino)]bis[*N,N'*-bis(2,3-dihydroxypropyl)-2,4,6-triiodoisophthalamide]; 1,3-bis(acetylamino)-*N,N'*-bis[3,5-bis(2,3-dihydroxypropylaminocarbonyl)-2,4,6-triiodophenyl]-2-hydroxypropane; 2-5410-3A; Acupaque; Visipaque. $C_{35}H_{44}I_6N_6O_{15}$; mol wt 1550.19. C 27.12%, H 2.86%, I 49.12%, N 5.42%, O 15.48%. Nonionic, dimeric x-ray contrast medium. Prepn: P. E. Hansen *et al.*, Eur. pat. Appl. **108,638** (1984 to Nyegaard), *C.A.* **101**, 151599g (1984). HPLC determn in plasma: H. Nomura *et al.*, *J. Chromatogr. Biomed. Appl.* **572**, 333 (1991). Clinical pharmacokinetics: M. G. Svaland *et al.*, *Invest. Radiol.* **27**, 130 (1992). Clinical studies in cerebral arteriography: Y. Palmers *et al.*, *Eur. J. Radiol.* **17**, 203 (1993); in cardiac angiography: J. A. H[illegible] *et al.*, *Am. J. Cardiol.* **74**, 57 (1994); in urography: [illegible] Conroy *et al.*, *Clin. Radiol.* **49**, 337 (1994). Use as density gradient medium: T. Ford *et al.*, *Anal. Biochem.* **220**, [illegible] (1994); J. Graham *et al.*, *ibid.* 367.



mp 240-250°. Soluble in water. Viscosity (37°) 11.[illegible] mPa·s; osmolality 290 mOsm/kg H_2O; pH 7.2-7.6 (c = 3[illegible] mgI/ml). 50% aqueous soln (w/v): viscosity 8.3 mPa·s; [illegible] 1.4128 ±0.0003; d 1.266 ±0.003; osmolality 200 ±5 mOsm/kg. LD_{50} i.v. in mice: 21 gI/kg (Palmers).
THERAP CAT: Diagnostic aid (radiopaque medium).

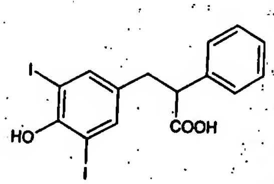
5045. Iodized Oil. Lipiodol. An iodine addition product of vegetable oils, contg 38-42% organically combined iodine.

Thick, viscous, oily liq; alliaceous odor; oleaginous taste; dec on exposure to air and light, becoming brown. *Keep in small, nearly full containers, protected from light.*
THERAP CAT: Diagnostic aid (radiopaque medium).
THERAP CAT (VET): X-ray contrast medium.

5046. Iodoacetic Acid. $C_2H_3IO_2$; mol wt 185.95. C 12.92%, H 1.63%, I 68.25%, O 17.21%. CH_2ICOOH. Prepd by treating chloroacetic acid in acetone soln with NaI. [illegible]

Colorless or white crystals. mp 82-83°. Sol in water, alc; very slightly sol in ether.

5047. Iodoalphionic Acid. *4-Hydroxy-3,5-diiodo-α-phenylbenzenepropanoic acid; β-(4-hydroxy-3,5-diiodophenyl)-α-phenylpropionic acid;* 3,5-diiodo-α-phenylphloretic acid; Coletrast; Priodax; Pheniodol; Dikol; Iodobil; Jodo[illegible]; Biliognost; Tenicid; Biliselectan. $C_{15}H_{12}I_2O_3$; mol wt 494.07. C 36.47%, H 2.45%, I 51.37%, O 9.71%. Prepd by iodinating β-(4-hydroxyphenyl)-α-phenylpropionic acid with iodine in an aq soln of alkali iodide in the presence of NH_3 or with iodine in acetic anhydride as the iodinating agent, *cf.* Brit. pat. **559,024** (1944), *C.A.* **40**, 1883 (1946). Prepn of optically active forms: Tullar, Hoppe, U.S. pat. **2,552,696** (1951 to Sterling Drug). Toxicity data: J. O. Hoppe, S. Archer, *Am. J. Roentgenol. Rad. Ther.* **69**, [illegible] (1953).



Consult the Name Index before using this section.

Weed Susceptibility Chart

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The herbicide ratings used in this chart are averages for the performance of these herbicides in the state when the herbicide is applied at labeled rates for crop production. Results may vary with location, rate of application, growth stage, and climatic conditions. Individuals are encouraged to alter the ratings to reflect their individual experiences with these herbicides under local conditions.

Ratings: "C" = weed is generally controlled, "P" = weed is injured or partially controlled, "N" = weed is not normally controlled, "-" = not sufficient information to rate the performance of this herbicide on the weed.

WEEDS		HERBICIDES		
COMMON NAME	BOTANICAL NAME[1]	COMMON NAME		TRADE NAME[2]
BROADLEAVED WEEDS				
BINDWEED(Pr)[4]	*Convolvulus arvensis*	ALACHLOR	=	LASSO
BINDWEED(Se)[3]	*Convolvulus arvensis*	ATRAZINE	=	AATREX
CARROT, WILD	*Daucus carota*	BENEFIN	=	BALAN
CHICKWEED	*Stellaria media*	BENSULIDE	=	BETASAN, PREFAR, PRESAN
CHINESE THORNAPPLE	*Datura ferox*	BENTAZON	=	BASAGRAN
CLOVER	*Medicago and Melilotus spp.*	BROMACIL	=	HYVAR
COCKLEBUR	*Xanthium strumarium*	BROMOXYNIL	=	BUCTRIL
CUDWEED	*Gnaphalium* spp.	CARFENTRAZONE	=	SHARK
CURLY DOCK(Pr)	*Rumex crispus*	CLETHODIM	=	PRISM, ENVOY
CURLY DOCK(Se)	*Rumex crispus*	CLOPYRALID	=	TRANSLINE, STINGER
DAISY, ENGLISH	*Bellis perennis*	CHLORSULFURON	=	GLEAN, TELAR
DANDELION(Pr)	*Taraxacum officinale*	CYCLOATE	=	RO-NEET
DANDELION(Se)	*Taraxacum officinale*	DAZOMET	=	BASAMID
DODDER	*Cuscuta* spp.	DCPA	=	DACTHAL
FIDDLENECK	*Amsinckia menziesii*	DICAMBA	=	BANVEL, CLARITY, VANQUISH
FILAREE	*Erodium* spp.	DICHLOBENIL	=	CASORON, NOROSAC
FLEABANE, HAIRY	*Conyza bonariensis*	DICLOFOP	=	HOELON
FLUVELLIN, SHARPPOINT	*Kickxia elatine*	DIFENZOQUAT	=	AVENGE
GOOSEFOOT, NETTLELEAF	*Chenopodium murale*	DIQUAT	=	REWARD
GROUNDCHERRY	*Physalis* spp.	DIMETHENAMID	=	OUTLOOK
GROUNDSEL, COMMON	*Senecio vulgarius*	DITHIOPYR	=	DIMENSION
HENBIT	*Lamium amplexicaule*	DIURON	=	KARMEX, DIREX
HORSETAIL, SCOURING RUSH	*Equisetum* spp.	DSMA	=	DSMA
HORSEWEED, MARESTAIL	*Conyza canadensis*	ENDOTHAL	=	AQUATHOL, ENDOTHAL, HYDROTHOL
KNOTWEED	*Polygonum* spp.	EPTC	=	EPTAM, ERADICANE
KOCHIA	*Kochia scoparia*	ETHAFLURALIN	=	CURBIT, SONALAN
KYLLINGA, GREEN	*Kyllinga brevifolia*	ETHOFUMESATE	=	NORTRON, PROGRASS
LAMBSQUARTERS, COMMON	*Chenopodium album*	FENOXAPROP	=	ACCLAIM, PUMA, WHIP
LONDON ROCKET	*Sisymbrium irio*	FLUAZIFOP	=	FUSILADE
MALLOW, CHEESEWEED	*Malva* spp.	FLUMIOXAZIN	=	CHATEAU
MINERS LETTUCE	*Claytonia perfoliata*	GLUFOSINATE	=	FINALE, LIBERTY, RELY
MORNINGGLORY, JAPANESE	*Ipomoea nil*	GLYPHOSATE	=	ROUNDUP[5], TOUCHDOWN[5], RODEO, OTHERS
MUSTARD	*Brassica spp.*	HALOSULFURON	=	MANAGE, PERMIT
NETTLE, BURNING	*Urtica urens*	HEXAZINONE	=	VELPAR, PRONONE
NIGHTSHADE, BLACK	*Solanum nigrum*	IMAZAMOX	=	RAPTOR
NIGHTSHADE, HAIRY	*Solanum sarrachoides = S. physalifolium*	IMAZAPIC	=	PLATEAU
NUTSEDGE, PURPLE	*Cyperus rotundus*	IMAZAPYR	=	ARSENAL, CHOPPER, HABITAT, STALKER
NUTSEDGE, YELLOW	*Cyperus esculentus*	IMAZETHAPYR	=	PURSUIT
ONION, WILD	*Allium canadense*	ISOXABEN	=	GALLERY
PEPPERWEED, PERENNIAL	*Lepidium latifolium*	LINURON	=	LOROX
PIGWEED	*Amaranthus spp.*	MCPA	=	MCPA

WEEDS		HERBICIDES		
COMMON NAME	BOTANICAL NAME[1]	COMMON NAME		TRADE NAME[2]
BROADLEAVED WEEDS				
PINEAPPLE WEED	*Matricaria matricarioides = Chamomilla suaveolens*	MECOPROP	=	MCPP
PLANTAIN, BUCKHORN (Pr)[4]	*Plantago lanceolata*	METHAM	=	VAPAM, SECTAGON
PLANTAIN, BUCKHORN (Se)[3]	*Plantago lanceolata*	METOLACHLOR	=	DUAL, PENNANT
PRICKLY LETTUCE	*Lactuca serriola*	METRIBUZIN	=	SENCOR
PUNCTUREVINE	*Tribulus terrestris*	MSMA	=	MSMA, BUENO 6
PURSLANE	*Portulaca oleracea*	NAPROPAMIDE	=	DEVRINOL
RADISH, WILD	*Raphanus* spp.	NICOSULFURON	=	ACCENT
REDMAIDS	*Calandrinia ciliata*	NORFLURAZON	=	SOLICAM, ZORIAL, PREDICT
RUSSIAN THISTLE	*Salsola tragus*	ORYZALIN	=	SURFLAN
SHEPHERD'S-PURSE	*Capsella bursa-pastoris*	OXADIAZON	=	RONSTAR
SMARTWEED	*Polygonium* spp.	OXYFLUORFEN	=	GOAL
SOWTHISTLE, ANNUAL	*Sonchus oleraceus*	PARAQUAT	=	GRAMOXONE
SPEEDWELL, PERSIAN	*Veronica persica*	PENDIMETHALIN	=	PROWL, PENDULUM, PRE-M
SPURGE, SPOTTED	*Euphobia maculata*	PELARGONIC ACID	=	SCYTHE
STARTHISTLE, YELLOW	*Centaurea solstitialis*	PHENMEDIPHAM	=	BETAMIX
SUNFLOWER, COMMON	*Helianthus annuus*	PRODIAMINE	=	BARRICADE, ENDURANCE
SWINECRESS	*Coronopus* spp.	PROMETRYN	=	CAPAROL
TANSY MUSTARD, FLIXWEED	*Descurainia* spp.,	PRONAMIDE	=	KERB
THISTLE, CANADA	*Cirsium arvense*	PYRAZON	=	PYRAMIN
TURKEY MULLEIN	*Eremocarpus setigerus*	PYRITHOBAC	=	STAPLE
VELVETLEAF	*Abutilon theophrasti*	RIMSULFURON	=	SHADEOUT, MATRIX
WILLOWHERB, PANICLE	*Epilobium brachycarpum*	SETHOXYDIM	=	POAST
WOODSORREL, CREEPING	*Oxalis corniculata*	SIMAZINE	=	PRINCEP
		THIAZOPYR	=	VISOR
GRASSES		TRICLOPYR	=	TURFLON,GARLON,REMEDY,GRANDSTAND, RENNOVATE
BARLEY, HARE	*Hordeum murinum*	TRIFLURALIN	=	TREFLAN, TRILIN
BARNYARDGRASS	*Echinochloa crus-galli*	2,4-D	=	2,4-D
BERMUDAGRASS(Pr)	*Cynodon dactylon*	2,4-DB	=	2,4-DB, BUTYRAC
BERMUDAGRASS(Se)	*Cynodon dactylon*			
BLUEGRASS, ANNUAL	*Poa annua*			
BLUEGRASS, BULBOUS	*Poa bulbosa*			
BROME, DOWNY; CHEATGRASS	*Bromus tectorum*			
BROME, RIPGUT	*Bromus rigidus*			
CANARYGRASS	*Phalaris spp.*			
CEREALS	*Volunteer cereals*			
CRABGRASS	*Digitaria spp.*			
DALLISGRASS(Pr)	*Paspalum dilatatum*			
DALLISGRASS(Se)	*Paspalum dilatatum*			
FALL PANICUM	*Panicum dichotomiflorum*			
FESCUE	*Festuca spp.*			
FOXTAIL, YELLOW	*Setaria pumila*			
GOOSEGRASS	*Eleusine indica*			
HARE BARLEY	*Hordeum leporinum*			
JOHNSONGRASS(Pr)	*Sorghum halepense*			
JOHNSONGRASS(Se)	*Sorghum halepense*			
OAT, WILD	*Avena fatua*			
RABBITSFOOT, POLYPOGON	*Polypogon monspeliensis*			
RESCUEGRASS	*Bromus catharticus*			
RYEGRASS, ITALIAN	*Lolium multiflorum*			
SANDBUR	*Cenchrus spp.*			
SPRANGLETOP, MEXICAN	*Leptochloa uninervia*			
STINKGRASS	*Eragrostis cilianensis*			

[1]COMMON AND BOTANICAL NAMES FROM: Weed Science Society of America http://www.wssa.net/weedname.html

[2] MOST COMMON NAMES USED, OTHER TRADENAMES POSSIBLE.

[3]Se = SEEDLING

[4]Pr = PERENNIAL

[5] GLYPHOSATE ISOPROPYLAMINE SALT

[6] GLYPHOSATE TRIMETHYLSULFONIUM SALT

Revised 11.10.06

Weed Susceptibility Chart

	ALACHLOR	ATRAZINE	BENEFIN	BENSULIDE	BENTAZON	BROMACIL	BROMOXYNIL	CARFENTRAZONE	CLETHODIM	CLOPYRALID
BROADLEAF										
BINDWEED(Pr)	N	N	N	N	N	P	N	P	N	N
BINDWEED(Se)	N	C	P	N	N	C	P	C	N	P
CARROT, WILD	-	-	-	-	-	C	-	-	N	P
CHICKWEED, COMMON	C	C	C	P	N	C	N	P	N	P
CLOVER	N	C	N	N	-	C	N	-	N	C
COCKLEBUR	-	C	N	N	C	C	P	P	N	C
CUDWEED	N	P	N	N	N	C	C	-	N	C
CURLY DOCK(Pr)	N	N	N	N	N	C	N	-	N	P
CURLY DOCK(Se)	C	C	P	N	N	C	C	-	N	C
DAISY, ENGLISH	-	-	-	N	-	-	N	N	N	-
DANDELION(Pr)	N	N	N	N	N	C	N	-	N	P
DANDELION(Se)	N	C	N	N	N	C	C	-	N	C
DODDER	N	N	N	N	N	N	N	P	N	N
FIDDLENECK	-	C	P	N	-	C	C	C	N	N
FILAREE	-	P	N	N	-	C	N	-	N	P
FLEABANE, HAIRY	N	C	N	N	N	C	C	N	N	-
FLUVELLIN	-	N	-	-	-	-	-	-	N	-
GOOSEFOOT, NETTLELEAF	P	C	C	P	N	C	C	-	N	N
GROUNDCHERRY	C	C	N	N	C	C	C	C	N	P
GROUNDSEL, COMMON	N	C	N	N	-	C	C	-	N	C
HENBIT	-	C	N	N	N	C	P	-	N	N
HORSETAIL	N	N	N	N	N	N	N	-	N	N
HORSEWEED, MARESTAIL	N	C	N	N	N	C	C	N	N	P
KNOTWEED	N	C	C	C	-	C	P	-	N	N
KOCHIA	P	C	P	P	P	C	C	-	N	C
KYLLINGA, GREEN	-	-	N	N	-	-	N	-	N	N
LAMBSQUARTERS	P	C	C	C	C	C	C	-	N	N
LONDON ROCKET	N	C	N	N	P	C	C	C	N	P
MALLOW, CHEESEWEED	P	C	N	N	C	C	P	C	N	N
MINERS LETTUCE	-	C	P	N	-	P	P	-	N	N
MORNINGGLORY, JAPANESE	N	C	N	N	P	C	C	C	N	N
MUSTARD	N	C	N	N	C	C	C	P	N	N
NETTLE, BURNING	C	C	N	P	-	C	P	C	N	-
NIGHTSHADE, BLACK	C	C	N	N	P	C	P	C	N	P
NIGHTSHADE, HAIRY	C	C	N	N	C	C	C	C	N	P
NUTSEDGE, PURPLE	N	N	N	N	N	C	N	N	N	N
NUTSEDGE, YELLOW	P	N	N	N	P	C	N	N	N	N
ONION, WILD	-	-	-	N	-	-	N	-	N	N
PEPPERWEED, PERENNIAL	N	N	N	N	N	-	N	N	N	N
PIGWEED	C	C	C	C	C	C	P	C	N	N
PINEAPPLE WEED	-	C	N	N	N	C	P	N	N	C
PLANTAIN, BUCKHORN(Pr)	-	P	N	N	N	P	N	-	N	N
PLANTAIN, BUCKHORN(Se)	-	C	N	N	N	C	C	-	N	N
PRICKLY LETTUCE	N	C	N	N	P	C	C	-	N	C
PUNCTUREVINE	-	P	P	N	N	C	C	-	N	N
PURSLANE	C	C	C	C	N	C	N	N	N	N
RADISH, WILD	N	C	N	N	P	C	C	P	N	N
REDMAIDS	-	-	-	-	-	-	-	-	-	N
RUSSIAN THISTLE	P	C	P	N	N	C	C	-	N	N
SHEPHERD'S-PURSE	N	C	N	N	-	C	C	P	N	P
SMARTWEED	P	C	P	N	N	C	P	-	N	P
SOWTHISTLE, ANNUAL	P	C	N	N	-	C	C	N	N	P
SPEEDWELL	-	-	-	-	-	-	-	N	-	N
SPURGE, SPOTTED	-	P	N	N	N	P	C	-	N	N
STARTHISTLE, YELLOW	-	C	N	N	-	C	P	N	N	C
SUNFLOWER, COMMON	N	C	N	N	C	C	C	-	N	C
SWINE CRESS	P	-	N	N	-	C	N	-	N	N
TANSY MUSTARD	-	C	N	N	-	C	C	-	N	N
THISTLE, CANADA	N	P	-	N	-	N	N	-	N	C
THORNAPPLE, CHINESE	N	-	N	N	-	C	C	-	N	P
TURKEY MULLEIN	-	N	N	N	N	P	N	-	N	N
VELVETLEAF	P	P	N	N	C	P	C	C	N	N
WILLOWHERB, PANICLE	-	-	N	N	-	C	-	-	N	N
WOODSORREL, CREEPING	N	-	N	N	N	C	N	-	N	N
GRASSES										
BARLEY, HARE	P	C	C	P	N	C	N	N	C	N
BARNYARDGRASS	C	C	C	C	N	C	N	N	C	N
BERMUDAGRASS(Pr)	N	N	N	N	N	C	N	N	P	N
BERMUDAGRASS(Se)	N	C	C	C	N	C	N	N	C	N
BLUEGRASS, ANNUAL	C	C	C	C	N	C	N	N	C	N
BLUEGRASS, BULBOUS	-	-	-	-	-	-	N	N	C	N
BROME, DOWNY	C	C	P	P	N	C	N	N	C	N
BROME, RIPGUT	-	P	P	-	N	C	N	N	C	N
CANARYGRASS	C	C	C	C	N	C	N	N	C	N
CEREALS, VOLUNTEER	N	C	P	N	N	C	N	N	C	N
CRABGRASS	C	P	C	C	N	C	N	N	C	N
DALLISGRASS(Pr)	N	N	N	N	N	C	N	N	N	N
DALLISGRASS(Se)	C	C	C	C	N	C	N	N	P	N
FESCUE	-	-	-	-	N	-	N	N	-	-
FOXTAIL, YELLOW	C	C	C	C	N	C	N	N	C	N
GOOSEGRASS	C	C	P	C	N	C	N	N	C	N
JOHNSONGRASS(Pr)	N	N	N	N	N	P	N	N	C	N
JOHNSONGRASS(Se)	C	C	C	C	N	C	N	N	C	N
OAT, WILD	N	C	P	N	N	C	N	N	C	N
PANICUM, FALL	C	C	C	C	N	C	N	N	-	N
RABBITFOOT, POLYPOGON	-	-	-	-	-	-	N	N	C	N
RESCUEGRASS	-	-	C	N	N	C	N	N	C	N
RYEGRASS, ITALIAN	C	C	C	P	N	C	N	N	C	N
SANDBUR	C	P	P	P	N	C	N	N	C	N
SPRANGLETOP, MEXICAN	C	C	P	C	N	C	N	N	C	N
STINKGRASS	C	C	C	C	N	C	N	N	C	N

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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Weed Susceptibility Chart

	CHLORSULFURON	CYCLOATE	DACTHAL	DAZOMET	DICAMBA	DICHLOBENIL	DICLOFOP	DIFENZOQUAT	DIQUAT	DIMETHENAMID
BROADLEAF										
BINDWEED(Pr)	-	N	N	P	P	P	N	N	N	N
BINDWEED(Se)	-	N	N	P	P	C	N	N	P	-
CARROT, WILD	-	-	N	-	C	-	N	-	N	-
CHICKWEED, COMMON	C	C	C	C	C	C	N	N	C	C
CLOVER	P	P	N	N	C	P	N	N	P	C
COCKLEBUR	-	N	N	C	C	P	N	N	P	-
CUDWEED	-	P	N	C	P	C	N	N	N	-
CURLY DOCK(Pr)	P	N	N	C	P	N	N	N	N	-
CURLY DOCK(Se)	C	P	C	C	C	C	N	N	C	-
DAISY, ENGLISH	-	-	-	-	-	N	N	-	-	-
DANDELION(Pr)	P	N	N	P	P	N	N	N	N	-
DANDELION(Se)	C	N	C	C	C	C	N	N	N	-
DODDER	-	N	C	N	N	C	N	N	N	-
FIDDLENECK	C	C	C	C	C	C	N	N	P	-
FILAREE	C	N	P	P	C	P	N	N	P	-
FLEABANE, HAIRY	-	C	N	C	C	P	N	N	C	
FLUVELLIN	-	N	N	-	-	-	-	-	-	
GOOSEFOOT, NETTLELEAF	-	C	C	C	C	C	N	N	C	
GROUNDCHERRY	-	C	C	C	C	C	N	N	C	C
GROUNDSEL, COMMON	C	C	N	C	C	C	N	N	C	
HENBIT	C	C	P	C	P	C	N	N	C	
HORSETAIL	C	N	N	-	N	C	N	N	N	-
HORSEWEED, MARESTAIL	C	C	N	C	C	P	N	N	P	
KNOTWEED	P	P	P	C	C	C	N	N	P	
KOCHIA	C	P	P	C	C	-	N	N	C	
KYLLINGA, GREEN	-	-	N	C	N	-	N	-	-	
LAMBSQUARTERS	C	C	C	C	C	C	N	N	C	C
LONDON ROCKET	C	N	P	C	C	C	N	N	C	
MALLOW, CHEESEWEED	C	P	P	P	P	C	N	N	N	-
MINERS LETTUCE	C	C	-	C	-	C	-	-	-	-
MORNINGGLORY, JAPANESE		N	N	-	C	C	N	N	P	-
MUSTARD	C	N	P	C	C	C	N	N	C	-
NETTLE, BURNING	-	P	P	C	P	C	N	N	P	-
NIGHTSHADE, BLACK	-	P	P	C	C	C	N	N	C	-
NIGHTSHADE, HAIRY	-	C	P	C	C	C	N	N	C	-
NUTSEDGE, PURPLE	-	P	N	P	N	P	N	N	N	-
NUTSEDGE, YELLOW	-	P	N	P	N	C	N	N	N	-
ONION, WILD	-	-	N	-	C	-	N	-	-	-
PEPPERWEED, PERENNIAL	C	N	N	C	P	N	N	N	N	-
PIGWEED	C	C	C	C	C	P	N	N	C	C
PINEAPPLE WEED	-	C	N	C	C	C	N	N	P	
PLANTAIN, BUCKHORN(Pr)	-	N	N	C	P	-	N	N	N	
PLANTAIN, BUCKHORN(Se)	-	N	N	C	C	C	N	N	C	
PRICKLY LETTUCE	C	C	N	C	C	C	N	N	P	
PUNCTUREVINE	C	P	P	C	C	C	N	N	C	
PURSLANE	-	C	C	C	C	C	N	N	C	C
RADISH, WILD	C	N	N	C	C	C	N	N	C	-
REDMAIDS	-	C	-	C	-	C	-	-	-	
RUSSIAN THISTLE	C	P	C	C	C	C	N	N	C	
SHEPHERD'S-PURSE	C	P	N	C	C	C	N	N	P	
SMARTWEED	-	P	C	C	C	-	N	N	-	
SOWTHISTLE, ANNUAL	C	C	P	C	C	C	N	N	P	-
SPEEDWELL	P	C	-	-	-	-	-	-	-	-
SPURGE, SPOTTED	-	C	P	C	P	C	N	N	C	-
STARTHISTLE, YELLOW	P	-	N	-	C	C	N	N	C	-
SUNFLOWER, COMMON	C	N	P	C	C	C	N	N	P	-
SWINE CRESS	C	-	N	C	P	-	N	N	P	-
TANSY MUSTARD	C	N	P	C	C	-	N	N	C	-
THISTLE, CANADA	P	-	N	P	P	-	N	-	-	N
THORNAPPLE, CHINESE	-	-	N	-	C	-	N	N	P	-
TURKEY MULLEIN	P	N	N	C	P	-	N	N	-	-
VELVETLEAF	-	N	N	-	C	-	N	N	P	N
WILLOWHERB, PANICLE	-	-	N	C	-	-	N	N	P	-
WOODSORREL, CREEPING	-	-	N	P	P	C	N	N	P	-
GRASSES										
BARLEY, HARE	N	C	C	C	N	C	N	N	P	C
BARNYARDGRASS	N	C	C	C	N	P	P	N	P	C
BERMUDAGRASS(Pr)	N	N	N	P	N	P	N	N	N	-
BERMUDAGRASS(Se)	-	C	C	C	N	P	N	N	P	-
BLUEGRASS, ANNUAL	-	C	C	C	N	C	-	N	P	C
BLUEGRASS, BULBOUS	N	-	-	-	N	-	-	-	-	-
BROME, DOWNY	N	C	C	C	N	C	C	N	P	-
BROME, RIPGUT	N	C	C	C	N	C	-	N	-	-
CANARYGRASS	N	C	C	C	N	C	P	N	P	-
CEREALS, VOLUNTEER	N	N	P	C	N	C	N	N	P	-
CRABGRASS	-	C	C	C	N	P	C	N	C	C
DALLISGRASS(Pr)	N	N	N	P	N	N	N	N	N	-
DALLISGRASS(Se)	-	C	C	C	P	C	N	N	N	C
FESCUE	P	C	-	-	N	-	-	-	-	-
FOXTAIL, YELLOW	P	C	C	C	N	C	C	N	C	C
GOOSEGRASS	-	C	C	C	N	C	N	N	P	-
JOHNSONGRASS(Pr)	N	N	N	P	N	N	N	N	N	-
JOHNSONGRASS(Se)	-	C	C	C	N	N	N	N	C	-
OAT, WILD	N	C	P	C	N	C	C	C	P	C
PANICUM, FALL	-	C	C	C	N	-	-	N	-	-
RABBITFOOT, POLYPOGON	N	-	-	-	N	-	-	-	P	-
RESCUEGRASS	-	C	N	C	N	-	-	N	-	-
RYEGRASS, ITALIAN	N	C	C	C	N	C	C	N	P	C
SANDBUR	-	C	C	C	N	C	C	N	P	-
SPRANGLETOP, MEXICAN	-	C	C	C	N	C	-	N	N	-
STINKGRASS	-	C	C	C	N	P	-	-	P	-

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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Weed Susceptibility Chart

	DITHIOPYR	DIURON	DSMA	ENDOTHAL	EPTC	ETHALFLURALIN	ETHOFUMESATE	FENOXAPROP	FLUAZIFOP	FLUMIOXAZIN
BROADLEAF										
BINDWEED(Pr)	N	N	N	N	N	-	N	N	N	N
BINDWEED(Se)	P	C	N	N	N	-	N	N	N	-
CARROT, WILD	-	P	-	-	N	-	-	N	N	-
CHICKWEED, COMMON	C	C	P	P	C	C	C	N	N	C
CLOVER	N	P	N	N	N	P	C	N	N	C
COCKLEBUR	-	C	P	P	N	-	P	N	N	-
CUDWEED	P	C	N	N	P	P	C	N	N	-
CURLY DOCK(Pr)	N	P	N	N	N	-	N	N	N	-
CURLY DOCK(Se)	-	C	N	C	C	C	-	N	N	-
DAISY, ENGLISH	N	N	-	-	-	-	-	N	N	N
DANDELION(Pr)	N	N	N	N	N	-	N	N	N	-
DANDELION(Se)	P	C	N	C	C	-	-	N	N	-
DODDER	C	N	N	N	N	-	N	-	N	-
FIDDLENECK	C	C	N	C	C	C	C	N	N	C
FILAREE	P	C	N	N	P	-	P	N	P	C
FLEABANE, HAIRY	N	P	N	P	C	-	P	N	N	C
FLUVELLIN	-	-	-	-	-	-	-	-	N	-
GOOSEFOOT, NETTLELEAF	C	C	N	N	C	-	C	N	N	C
GROUNDCHERRY	C	C	N	P	C	-	C	N	N	C
GROUNDSEL, COMMON	P	N	N	N	C	C	P	N	N	C
HENBIT	C	C	C	P	C	P	P	N	N	C
HORSETAIL	N	N	N	N	N	-	N	N	N	N
HORSEWEED, MARESTAIL	N	P	N	N	C	-	P	N	N	C
KNOTWEED	C	C	N	C	P	P	C	N	N	C
KOCHIA	-	P	-	-	P	-	P	N	N	-
KYLLINGA, GREEN	N	-	P	N	P	N	-	N	N	N
LAMBSQUARTERS	P	C	N	P	C	-	C	N	N	C
LONDON ROCKET	C	C	N	N	C	N	N	N	N	-
MALLOW, CHEESEWEED	P	P	N	N	N	P	P	N	N	C
MINERS LETTUCE	C	C	-	-	N	C	-	-	N	C
MORNINGGLORY, JAPANESE	-	C	P	-	-	-	C	N	N	C
MUSTARD	C	C	N	P	N	P	N	N	N	C
NETTLE, BURNING	C	C	N	C	C	P	P	N	N	C
NIGHTSHADE, BLACK	C	C	N	N	P	P	P	N	N	C
NIGHTSHADE, HAIRY	C	C	N	P	C	P	C	N	N	C
NUTSEDGE, PURPLE	N	N	P	N	P	-	P	N	N	N
NUTSEDGE, YELLOW	N	N	P	N	P	-	P	N	N	P
ONION, WILD	-	-	-	-	N	-	-	N	N	-
PEPPERWEED, PERENNIAL	N	N	N	N	N	-	N	N	N	N
PIGWEED	P	C	N	N	C	C	P	N	N	C
PINEAPPLE WEED	C	C	N	-	C	P	P	N	N	C
PLANTAIN, BUCKHORN(Pr)	N	N	N	N	-	-	N	N	N	-
PLANTAIN, BUCKHORN(Se)	-	C	N	-	-	-	N	N	N	-
PRICKLY LETTUCE	-	C	N	P	C	C	C	N	N	C
PUNCTUREVINE	-	P	P	N	N	N	C	N	N	-
PURSLANE	C	C	N	N	C	C	C	N	N	C
RADISH, WILD	-	C	N	N	N	-	N	N	N	-
REDMAIDS	C	-	-	-	-	C	-	-	N	C
RUSSIAN THISTLE	-	P	N	N	P	-	P	N	N	-
SHEPHERD'S-PURSE	C	C	N	P	P	P	N	N	N	C
SMARTWEED	-	C	-	P	C	-	N	N	N	-
SOWTHISTLE, ANNUAL	P	C	N	P	C	C	C	N	N	C
SPEEDWELL	-	-	-	-	-	C	-	-	N	-
SPURGE, SPOTTED	C	N	N	N	N	-	N	N	N	-
STARTHISTLE, YELLOW	N	C	-	-	-	-	-	N	N	C
SUNFLOWER, COMMON	N	C	N	N	P	-	N	N	N	-
SWINE CRESS	-	C	-	N	N	-	N	N	N	-
TANSY MUSTARD	-	C	N	P	N	-	-	N	N	-
THISTLE, CANADA	N	N	-	-	N	-	-	N	N	-
THORNAPPLE, CHINESE	N	-	N	-	N	-	N	N	N	-
TURKEY MULLEIN	-	N	N	N	N	-	-	N	N	-
VELVETLEAF	C	-	-	-	-	-	-	N	N	-
WILLOWHERB, PANICLE	P	N	N	-	-	-	-	N	N	C
WOODSORREL, CREEPING	C	C	P	N	-	-	N	N	N	-
GRASSES										
BARLEY, HARE	C	C	N	N	C	C	C	-	C	-
BARNYARDGRASS	C	C	P	N	C	C	P	C	C	-
BERMUDAGRASS(Pr)	N	N	N	N	N	-	N	P	P	-
BERMUDAGRASS(Se)	C	N	N	N	C	-	-	P	C	-
BLUEGRASS, ANNUAL	C	C	N	C	C	C	C	N	N	-
BLUEGRASS, BULBOUS	-	N	-	-	-	-	-	-	-	-
BROME, DOWNY	C	C	N	N	C	C	-	-	C	-
BROME, RIPGUT	C	C	-	N	C	C	C	-	C	-
CANARYGRASS	C	C	N	P	C	C	C	C	C	-
CEREALS, VOLUNTEER	P	C	N	P	C	C	C	N	C	-
CRABGRASS	C	C	C	N	C	-	P	C	C	P
DALLISGRASS(Pr)	N	N	C	N	N	-	N	N	N	-
DALLISGRASS(Se)	C	C	C	N	C	-	-	N	-	-
FESCUE	C	-	-	-	-	C	-	-	-	-
FOXTAIL, YELLOW	C	C	-	N	C	-	C	C	C	-
GOOSEGRASS	P	C	C	N	C	C	N	P	C	
JOHNSONGRASS(Pr)	N	N	N	N	N	-	N	N	C	-
JOHNSONGRASS(Se)	C	C	C	N	C	-	C	C	C	-
OAT, WILD	P	P	N	N	C	C	C	C	C	C
PANICUM, FALL	-	N	C	N	C	-	-	-	-	-
RABBITFOOT, POLYPOGON	-	C	-	-	C	-	-	C	C	-
RESCUEGRASS	C	-	-	N	C	C	N	-	-	-
RYEGRASS, ITALIAN	C	C	N	N	C	N	N	N	C	-
SANDBUR	C	C	N	N	C	-	-	-	C	-
SPRANGLETOP, MEXICAN	C	N	N	N	C	-	-	N	P	-
STINKGRASS	C	C	-	N	C	-	C	-	C	-

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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Weed Susceptibility Chart

	GLUFOSINATE	GLYPHOSATE	HALOSULFURON	HEXAZINONE	IMAZAMOX	IMAZAPIC	IMAZAPYR	IMAZETHAPYR	ISOXABEN
BROADLEAF									
BINDWEED(Pr)	P	P	N	N	P	-	C	N	C
BINDWEED(Se)	C	C	-	C	P	-	-	-	C
CARROT, WILD	-	C	-	-	-	-	-	-	-
CHICKWEED, COMMON	C	C	C	C	C	-	C	P	C
CLOVER	C	P	-	C	N	N	P	N	C
COCKLEBUR	C	C	C	C	P	N	C	P	-
CUDWEED	C	C	-	C	-	N	C	P	C
CURLY DOCK(Pr)	-	P	-	N	N	-	C	P	N
CURLY DOCK(Se)	C	C	-	C	N	-	-	P	C
DAISY, ENGLISH	-	C	-	-	-	N	-	-	-
DANDELION(Pr)	-	C	-	C	P	N	-	P	C
DANDELION(Se)	C	C	-	C	P	N	C	P	C
DODDER	P	C	-	N	P	-	-	P	C
FIDDLENECK	C	C	-	C	P	-	C	P	C
FILAREE	C	P	-	P	C	C	C	P	C
FLEABANE, HAIRY	C	C	-	C	-	N	C	N	C
FLUVELLIN	-	P	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	C	C	-	C	-	-	C	P	C
GROUNDCHERRY	C	C	-	C	-	-	-	C	C
GROUNDSEL, COMMON	C	C	C	C	-	N	-	P	C
HENBIT	C	C	-	P	P	-	C	P	C
HORSETAIL	N	P	N	N	-	-	-	N	N
HORSEWEED, MARESTAIL	C	C	-	C	-	N	C	N	C
KNOTWEED	P	P	-	C	P	-	C	C	C
KOCHIA	C	C	P	C	P	-	C	P	-
KYLLINGA, GREEN	-	N	P	-	-	-	-	-	N
LAMBSQUARTERS	C	C	N	C	C	-	C	P	C
LONDON ROCKET	C	C	C	C	P	C	C	C	C
MALLOW, CHEESEWEED	C	P	C	P	C	-	P	C	C
MINERS LETTUCE	-	C	-	C	C	-	-	C	C
MORNINGGLORY, JAPANESE	C	P	P	C	P	-	C	P	C
MUSTARD	C	C	C	P	C	C	-	C	C
NETTLE, BURNING	C	N	C	C	P	-	C	C	C
NIGHTSHADE, BLACK	C	C	N	C	C	-	-	C	C
NIGHTSHADE, HAIRY	C	C	N	C	C	-	-	C	C
NUTSEDGE, PURPLE	P	P	C	N	-	-	-	P	N
NUTSEDGE, YELLOW	P	P	C	N	-	-	-	P	N
ONION, WILD	-	-	-	-	-	-	-	-	-
PEPPERWEED, PERENNIAL	-	P	N	-	P	C	C	P	N
PIGWEED	C	C	P	C	C	-	C	C	C
PINEAPPLE WEED	C	C	-	P	-	N	-	-	-
PLANTAIN, BUCKHORN(Pr)	-	P	-	P	N	-	-	-	N
PLANTAIN, BUCKHORN(Se)	C	C	-	C	P	-	C	-	-
PRICKLY LETTUCE	C	C	C	C	N	N	C	N	C
PUNCTUREVINE	C	C	-	C	P	-	C	C	C
PURSLANE	C	P	P	C	P	-	C	C	C
RADISH, WILD	C	C	C	C	C	C	C	P	C
REDMAIDS	-	C	-	-	-	-	-	-	-
RUSSIAN THISTLE	C	C	-	C	P	-	C	P	C
SHEPHERD'S-PURSE	C	C	C	C	C	C	C	C	C
SMARTWEED	C	C	P	-	P		C	C	-
SOWTHISTLE, ANNUAL	C	C	C	C	P	N	P	N	C
SPEEDWELL	-	C	-	-	-	-	-	-	-
SPURGE, SPOTTED	C	C	-	C	P	-	C	P	C
STARTHISTLE, YELLOW	C	C	-	C	N	N	C	P	-
SUNFLOWER, COMMON	C	C	C	C	C	N	C	P	C
SWINE CRESS	-	C	N	C	C	C	C	C	-
TANSY MUSTARD	C	C	-	C	C	C	C	C	-
THISTLE, CANADA	-	P	N	N	P	N	-	-	N
THORNAPPLE, CHINESE	-	C	-	-	C	-	-	-	-
TURKEY MULLEIN	C	P	-	P	-	-	-	-	-
VELVETLEAF	-	P	C	-	C	-	C	C	C
WILLOWHERB, PANICLE	C	P	-	-	-	-	C	C	P
WOODSORREL, CREEPING	-	C	-	P	-	-	C	-	C
GRASSES									
BARLEY, HARE	-	C	-	P	C	C	-	-	N
BARNYARDGRASS	C	C	P	C	P	-	C	P	N
BERMUDAGRASS(Pr)	P	C	N	N	-	-	-	N	N
BERMUDAGRASS(Se)	C	C	N	P	-	-	P	P	N
BLUEGRASS, ANNUAL	C	C	N	P	P	C	C	N	N
BLUEGRASS, BULBOUS	-	C	-	-	C	C	C	-	-
BROME, DOWNY	C	C	-	C	C	C	C	N	N
BROME, RIPGUT	C	C	-	P	C	C	C	N	N
CANARYGRASS	C	C	-	C	P	C	C	P	N
CEREALS, VOLUNTEER	C	C	-	P	C	C	-	P	N
CRABGRASS	C	C	C	C	P	-	C	P	N
DALLISGRASS(Pr)	P	C	N	P	-	-	-	N	N
DALLISGRASS(Se)	C	C	N	C	-	-	P	-	N
FESCUE	-	C	-	-	-	P	-	-	-
FOXTAIL, YELLOW	C	C	-	C	C	-	C	C	N
GOOSEGRASS	-	C	-	-	-	-	C	-	N
JOHNSONGRASS(Pr)	P	C	-	N	N	-	-	P	N
JOHNSONGRASS(Se)	C	C	-	C	P	-	C	C	N
OAT, WILD	C	C	-	P	C	P	C	P	N
PANICUM, FALL	-	C	-	C	-	-	C	P	N
RABBITFOOT, POLYPOGON	-	C		-	-	-	-	-	-
RESCUEGRASS	-	C	-	P	C	C	-	N	N
RYEGRASS, ITALIAN	-	C	N	C	C	C	C	P	N
SANDBUR	C	C	-	C	C	-	C	P	N
SPRANGLETOP, MEXICAN	C	C	-	-	-	-	P	N	N
STINKGRASS	C	C	-	-	-	-	C	-	N

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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Weed Susceptibility Chart

	LINURON	MCPA	MECOPROP	METHAM	METOLACHLOR	METRIBUZIN	MSMA	NAPROPAMIDE	NICOSULFURON	NORFLURAZON
BROADLEAF										
BINDWEED(Pr)	N	N	N	P	N	N	N	N	-	N
BINDWEED(Se)	N	N	N	P	N	N	N	N	-	P
CARROT, WILD	N	-	-	-	N	-	-	-	-	-
CHICKWEED, COMMON	C	P	P	C	C	C	P	C	-	P
CLOVER	P	N	P	N	N	N	N	P	-	N
COCKLEBUR	C	C	C	C	-	P	P	C	N	C
CUDWEED	P	P	P	C	N	-	N	P	-	P
CURLY DOCK(Pr)	N	P	P	C	N	N	N	N	-	N
CURLY DOCK(Se)	C	C	C	C	C	C	N	P	-	N
DAISY, ENGLISH	-	-	-	-	-	-	-	-	-	-
DANDELION(Pr)	N	N	N	P	N	N	N	N	-	N
DANDELION(Se)	N	C	C	C	N	C	N	C	-	N
DODDER	N	N	N	N	N	N	N	N	-	N
FIDDLENECK	C	P	P	C	N	C	N	C	-	P
FILAREE	C	P	-	C	N	C	N	C	-	P
FLEABANE, HAIRY	N	C	C	C	N	C	N	N	-	P
FLUVELLIN	-	-	-	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	C	C	C	C	P	C	N	C	-	C
GROUNDCHERRY	C	C	C	C	C	P	N	N	-	C
GROUNDSEL, COMMON	C	N	N	C	N	P	N	C	-	P
HENBIT	C	N	N	C	-	C	C	N	-	P
HORSETAIL	N	N	N	-	N	N	N	N	-	N
HORSEWEED, MARESTAIL	N	C	C	C	N	P	N	N	-	P
KNOTWEED	P	P	P	C	N	-	N	C	-	P
KOCHIA	C	-	-	C	P	C	-	C	P	-
KYLLINGA, GREEN	N	-	-	-	N	-	P	N	-	P
LAMBSQUARTERS	C	C	C	C	P	C	N	C	P	P
LONDON ROCKET	C	C	C	C	N	C	N	C	-	P
MALLOW, CHEESEWEED	C	P	P	N	P	C	N	P	-	P
MINERS LETTUCE	-	-	-	C	-	-	-	-	-	P
MORNINGGLORY, JAPANESE	P	P	P	P	N	N	P	P	P	C
MUSTARD	C	C	C	C	N	C	N	P	-	P
NETTLE, BURNING	C	P	C	C	C	C	N	P	-	C
NIGHTSHADE, BLACK	C	C	C	C	C	P	N	N	N	C
NIGHTSHADE, HAIRY	P	C	C	C	C	N	N	N	-	C
NUTSEDGE, PURPLE	N	N	N	P	N	N	P	N	-	P
NUTSEDGE, YELLOW	P	N	N	C	P	P	C	N	N	P
ONION, WILD	-	-	-	-	-	-	-	-	-	-
PEPPERWEED, PERENNIAL	N	N	N	C	N	N	N	N	-	N
PIGWEED	C	C	C	C	C	C	N	C	C	P
PINEAPPLE WEED	C	P	N	C	-	P	N	P	-	P
PLANTAIN, BUCKHORN(Pr)	-	P	P	C	N	N	N	N	-	N
PLANTAIN, BUCKHORN(Se)	-	C	C	C	-	N	N	-	-	-
PRICKLY LETTUCE	C	C	C	C	N	C	N	C	-	P
PUNCTUREVINE	C	C	C	C	-	-	P	P	-	C
PURSLANE	C	P	P	C	C	P	N	C	-	C
RADISH, WILD	C	C	C	C	N	C	N	N	-	N
REDMAIDS	-	-	-	C	-	-	-	-	-	-
RUSSIAN THISTLE	P	P	P	C	P	-	N	C	-	C
SHEPHERD'S-PURSE	C	C	C	C	P	C	N	P	-	P
SMARTWEED	-	P	P	C	P	-	-	-	C	-
SOWTHISTLE, ANNUAL	C	C	C	C	P	C	N	C	-	P
SPEEDWELL	-	-	-	-	-	-	-	-	-	-
SPURGE, SPOTTED	N	P	P	C	N	C	N	N	-	P
STARTHISTLE, YELLOW	C	P	P	-	-	C	N	-	-	-
SUNFLOWER, COMMON	C	C	C	C	N	P	N	P	-	N
SWINE CRESS	C	N	N	C	P	C	-	C	-	-
TANSY MUSTARD	C	C	C	C	-	C	N	-	-	P
THISTLE, CANADA	-	-	-	P	N	-	-	-	-	-
THORNAPPLE, CHINESE	-	-	-	C	N	-	N	N	-	C
TURKEY MULLEIN	-	N	N	C	-	-	N	C	-	P
VELVETLEAF	-	-	-	-	P	-	-	-	P	-
WILLOWHERB, PANICLE	-	P	P	C	-	-	-	N	-	P
WOODSORREL, CREEPING	C	P	P	P	N	-	P	N	-	-
GRASSES										
BARLEY, HARE	C	N	N	C	P	C	N	C	-	C
BARNYARDGRASS	C	N	N	C	C	P	P	C	C	C
BERMUDAGRASS(Pr)	N	N	N	P	N	N	N	N	-	P
BERMUDAGRASS(Se)	N	N	N	C	N	N	N	C	-	C
BLUEGRASS, ANNUAL	C	N	N	C	C	P	N	C	-	C
BLUEGRASS, BULBOUS	-	N	N	-	-	-	-	-	-	-
BROME, DOWNY	-	N	N	C	C	C	-	C	-	C
BROME, RIPGUT	-	N	N	C	-	-	-	C	-	C
CANARYGRASS	C	N	N	C	C	C	N	C	-	C
CEREALS, VOLUNTEER	P	N	N	C	N	P	N	C	-	C
CRABGRASS	C	N	N	C	C	C	C	C	P	C
DALLISGRASS(Pr)	N	N	N	P	N	N	C	N	-	N
DALLISGRASS(Se)	N	N	N	C	C	C	C	C	-	N
FESCUE	-	N	N	C	-	-	-	-	-	-
FOXTAIL, YELLOW	C	N	N	C	C	C	-	C	C	C
GOOSEGRASS	C	N	N	C	C	P	C	-	-	C
JOHNSONGRASS(Pr)	N	N	N	P	N	N	N	N	C	C
JOHNSONGRASS(Se)	N	N	N	C	C	N	C	C	C	C
OAT, WILD	C	N	N	C	N	P	N	C	-	C
PANICUM, FALL	C	N	N	C	C	C	C	C	-	C
RABBITFOOT, POLYPOGON	C	N	N	-	C	-	-	-	-	C
RESCUEGRASS	-	N	N	C	-	-	-	-	-	-
RYEGRASS, ITALIAN	N	N	N	C	C	P	N	C	-	C
SANDBUR	-	N	N	C	C	C	C	C	-	C
SPRANGLETOP, MEXICAN	N	N	N	C	C	N	N	C	-	C
STINKGRASS	C	N	N	C	C	P	-	C	-	C

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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Weed Susceptibility Chart

	ORYZALIN	OXADIAZON	OXYFLUORFEN	PARAQUAT	PELARGONIC ACID	PENDIMETHALIN	PHENMEDIPHAM	PRODIAMINE	PROMETRYN
BROADLEAF									
BINDWEED(Pr)	N	P	N	N	N	N	N	P	N
BINDWEED(Se)	P	C	N	P	-	P	P	P	P
CARROT, WILD	N	-	-	P	-	-	-	N	N
CHICKWEED, COMMON	C	N	N	C	C	C	C	C	C
CLOVER	N	N	P	P	-	N	P	N	P
COCKLEBUR	N	-	C	C	-	N	C	N	C
CUDWEED	N	C	N	N	-	N	P	N	P
CURLY DOCK(Pr)	N	N	N	N	N	N	N	N	N
CURLY DOCK(Se)	P	-	C	C	C	C	C	C	C
DAISY, ENGLISH	N	-	-	N	-	-	-	N	N
DANDELION(Pr)	N	N	N	N	N	N	N	N	N
DANDELION(Se)	N	-	C	N	C	N	C	N	C
DODDER	N	-	N	N	-	P	N	C	N
FIDDLENECK	C	C	C	P	C	C	C	C	C
FILAREE	P	C	C	P	P	N	P	N	C
FLEABANE, HAIRY	N	P	P	P	P	N	C	N	C
FLUVELLIN	-	-	-	-	-	-	-	-	-
GOOSEFOOT, NETTLELEAF	C	C	C	C	-	C	C	C	C
GROUNDCHERRY	N	C	C	C	-	N	C	N	C
GROUNDSEL, COMMON	N	C	C	C	C	N	C	N	C
HENBIT	C	C	C	C	-	C	C	C	C
HORSETAIL	N	N	N	N	N	N	N	N	N
HORSEWEED, MARESTAIL	N	P	P	P	-	N	C	N	C
KNOTWEED	C	C	P	P	C	C	P	C	C
KOCHIA	C	-	P	C	-	C	P	C	C
KYLLINGA, GREEN	N	N	N	-	-	N	-	N	N
LAMBSQUARTERS	C	C	C	C	P	C	C	C	C
LONDON ROCKET	N	C	P	C	C	P	C	N	C
MALLOW, CHEESEWEED	P	C	C	P	C	P	P	N	C
MINERS LETTUCE	-	-	-	C	-	-	-	-	-
MORNINGGLORY, JAPANESE	P	P	C	P	-	N	P	N	C
MUSTARD	N	C	C	C	C	P	C	N	C
NETTLE, BURNING	P	C	C	P	-	N	C	N	C
NIGHTSHADE, BLACK	N	C	C	C	-	N	C	N	C
NIGHTSHADE, HAIRY	N	C	C	C	-	N	C	N	C
NUTSEDGE, PURPLE	N	N	N	N	N	N	N	N	N
NUTSEDGE, YELLOW	N	N	N	N	N	N	N	N	N
ONION, WILD	N	-	N	-	-	-	-	N	N
PEPPERWEED, PERENNIAL	N	N	N	N	N	N	N	N	N
PIGWEED	C	C	C	C	P	C	C	C	C
PINEAPPLE WEED	N	C	P	P	-	N	P	P	C
PLANTAIN, BUCKHORN(Pr)	N	N	N	N	N	N	N	N	N
PLANTAIN, BUCKHORN(Se)	-	-	-	C	C	-	-	-	C
PRICKLY LETTUCE	N	C	C	P	-	N	C	N	C
PUNCTUREVINE	P	-	C	C	-	P	P	P	P
PURSLANE	C	C	C	C	C	C	C	C	C
RADISH, WILD	N	C	C	C	-	N	C	N	C
REDMAIDS	-	-	-	C	-	-	-	-	-
RUSSIAN THISTLE	P	-	P	C	C	P	N	P	P
SHEPHERD'S-PURSE	N	C	P	P	C	P	C	N	C
SMARTWEED	-	-	-	P	-	-	P	-	-
SOWTHISTLE, ANNUAL	N	C	C	P	-	N	C	N	C
SPEEDWELL	-	-	-	C	-	-	-	-	-
SPURGE, SPOTTED	C	C	N	C	-	P	C	P	-
STARTHISTLE, YELLOW	N	-	C	C	-	N	-	-	C
SUNFLOWER, COMMON	N	-	C	P	-	N	P	N	C
SWINE CRESS	-	-	N	C	-	P	N	-	C
TANSY MUSTARD	N	-	-	P	-	N	C	N	C
THISTLE, CANADA	N	-	-	N	-	-	-	N	N
THORNAPPLE, CHINESE	N	-	P	P	-	N	-	-	P
TURKEY MULLEIN	N	-	P	P	-	N	-	N	-
VELVETLEAF	N	C	-	C	C	N	N	N	-
WILLOWHERB, PANICLE	P	-	C	N	-	-	-	N	-
WOODSORREL, CREEPING	P	C	C	P	P	P	-	P	-
GRASSES									
BARLEY, HARE	C	C	P	P	-	C	N	C	C
BARNYARDGRASS	C	C	P	P	C	C	N	C	P
BERMUDAGRASS(Pr)	N	N	N	N	N	N	N	N	N
BERMUDAGRASS(Se)	C	-	N	P	-	C	N	C	P
BLUEGRASS, ANNUAL	C	C	P	P	C	C	N	C	C
BLUEGRASS, BULBOUS	N	-	-	P	-	-	N	C	N
BROME, DOWNY	C	-	N	P	-	C	N	C	-
BROME, RIPGUT	C	C	-	-	-	C	N	C	P
CANARYGRASS	C	-	P	P	-	C	N	C	C
CEREALS, VOLUNTEER	C	-	N	P	-	C	N	C	P
CRABGRASS	C	C	N	C	-	C	N	C	P
DALLISGRASS(Pr)	N	N	N	N	N	N	N	N	N
DALLISGRASS(Se)	C	-	N	N	-	C	N	C	C
FESCUE	-	-	-	P	-	-	N	-	-
FOXTAIL, YELLOW	C	C	N	C	-	C	N	C	-
GOOSEGRASS	C	C	-	P	-	C	N	C	-
JOHNSONGRASS(Pr)	N	N	N	N	N	N	N	N	N
JOHNSONGRASS(Se)	C	-	N	C	-	C	N	C	N
OAT, WILD	C	C	P	P	-	P	N	P	C
PANICUM, FALL	C	C	N	N	-	C	N	C	-
RABBITFOOT, POLYPOGON	C	-	-	C	C	C	N	C	N
RESCUEGRASS	C	-	-	-	-	C	N	C	-
RYEGRASS, ITALIAN	C	N	N	P	-	C	N	C	P
SANDBUR	P	-	N	P	-	C	N	C	-
SPRANGLETOP, MEXICAN	C	-	N	N	N	C	N	C	N
STINKGRASS	C	-	C	P	-	C	N	C	P

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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Weed Susceptibility Chart

	PRONAMIDE	PYRAZON	PYRITHOBAC	RIMSULFURON	SETHOXYDIM	SIMAZINE	SULFENTRAZONE	THIAZOPYR	TRICLOPYR	TRIFLURALIN
BROADLEAF										
BINDWEED(Pr)	N	N	N	P	N	N		P	P	P
BINDWEED(Se)	N	N	P	P	N	C		C	C	P
CARROT, WILD	N	-	-	-	N	-		-	C	N
CHICKWEED, COMMON	C	C	N	C	N	C		P	C	C
CLOVER	N	N	N	-	N	C		-	C	N
COCKLEBUR	N	P	C	P	N	C		-	-	N
CUDWEED	N	C	N	C	N	N		C	-	N
CURLY DOCK(Pr)	N	N	N	-	N	N		N	P	N
CURLY DOCK(Se)	C	C	N	C	N	C		-	C	C
DAISY, ENGLISH	N	-	-	-	N	-		-	-	N
DANDELION(Pr)	N	N	N	N	N	N		-	C	N
DANDELION(Se)	N	C	N	P	N	C		-	C	N
DODDER	P	N	N	C	N	N		C	N	C
FIDDLENECK	C	C	N	C	-	C		C	-	C
FILAREE	N	P	N	C	N	C		C	P	N
FLEABANE, HAIRY	N	C	N	C	N	C		C	-	N
FLUVELLIN	-	-	-	-	N	-		-	-	-
GOOSEFOOT, NETTLELEAF	C	C	C	-	N	C		-	C	C
GROUNDCHERRY	C	C	P	-	N	C		-	C	N
GROUNDSEL, COMMON	N	C	N	C	N	P[1]		C	C	N
HENBIT	C	C	N	C	N	C		-	-	C
HORSETAIL	N	N	N	-	N	N		N	N	N
HORSEWEED, MARESTAIL	N	C	N	-	N	C		C	C	N
KNOTWEED	C	P	C	C	N	C		C	-	C
KOCHIA	C	C	-	C	N	C		-	-	-
KYLLINGA, GREEN	N	-	-	-	N	-		-	-	N
LAMBSQUARTERS	C	C	N	P	N	C		P	C	C
LONDON ROCKET	C	C	C	C	N	C		P	C	N
MALLOW, CHEESEWEED	P	P	N	C	N	P		C	N	N
MINERS LETTUCE	P	C	-	-	N	-		-	-	C
MORNINGGLORY, JAPANESE	-	P	P	P	N	C		-	-	N
MUSTARD	C	C	C	C	N	C		P	C	N
NETTLE, BURNING	C	C	P	-	N	C		C	-	N
NIGHTSHADE, BLACK	C	C	C	P	N	C		P	C	N
NIGHTSHADE, HAIRY	C	C	C	P	N	C		-	C	N
NUTSEDGE, PURPLE	N	N	N	-	N	N		P	N	N
NUTSEDGE, YELLOW	N	N	N	P	N	N		P	N	N
ONION, WILD	-	-	-	-	N	-		-	P	N
PEPPERWEED, PERENNIAL	N	N	N	-	N	N		N	N	N
PIGWEED	C	P	C	C	N	C		P	C	C
PINEAPPLE WEED	N	P	-	C	N	C		-	-	N
PLANTAIN, BUCKHORN(Pr)	N	N	-	C	N	N		-	P	N
PLANTAIN, BUCKHORN(Se)	N	-	-	C	N	C		-	C	-
PRICKLY LETTUCE	N	C	N	C	N	C		C	C	N
PUNCTUREVINE	N	P	P	C	N	P		-	-	P
PURSLANE	C	C	P	P	N	C		C	-	C
RADISH, WILD	P	C	N	C	N	P		-	C	N
REDMAIDS	-	C	-	-	N	-		C	-	-
RUSSIAN THISTLE	P	P	N	P	N	C		-	P	P
SHEPHERD'S-PURSE	C	C	C	C	N	C		C	C	N
SMARTWEED	-	-	-	P	N	C		-	-	-
SOWTHISTLE, ANNUAL	N	C	N	C	N	C		C	C	N
SPEEDWELL	-	C	-	-	N	-		-	-	-
SPURGE, SPOTTED	P	C	N	-	N	N		P	P	P
STARTHISTLE, YELLOW	N	-	-	N	N	C		-	P	N
SUNFLOWER, COMMON	N	P	C	C	N	C		-	C	N
SWINE CRESS	N	C	-	-	N	C		P	-	N
TANSY MUSTARD	C	C	-	C	N	C		-	-	N
THISTLE, CANADA	N	-	-	-	N	N		-	P	N
THORNAPPLE, CHINESE	N	-	P	-	N	-		-	-	N
TURKEY MULLEIN	N	-	-	-	N	N		-	-	N
VELVETLEAF	-	-	C	P	N	-		C	-	N
WILLOWHERB, PANICLE	-	-	-	-	N	N		-	-	-
WOODSORREL, CREEPING	-	N	N	-	N	C		C	C	P
GRASSES										
BARLEY, HARE	C	N	N	C	P	C		C	N	C
BARNYARDGRASS	C	N	N	C	C	P		C	N	C
BERMUDAGRASS(Pr)	N	N	N	N	P	N		N	P	N
BERMUDAGRASS(Se)	C	N	N	-	C	P		C	P	C
BLUEGRASS, ANNUAL	C	N	N	C	N	C		C	N	C
BLUEGRASS, BULBOUS	C	N	N	-	N	C		C	N	P
BROME, DOWNY	C	N	N	-	P	C		C	N	C
BROME, RIPGUT	C	N	N	C	C	-		-	N	C
CANARYGRASS	C	N	N	-	C	C		C	N	C
CEREALS, VOLUNTEER	C	N	N	C	C	C		C	N	P
CRABGRASS	C	N	N	P	C	N		C	N	C
DALLISGRASS(Pr)	N	N	N	-	-	N		-	N	N
DALLISGRASS(Se)	C	N	N	-	C	C		-	N	C
FESCUE	-	N	N	-	-	-		-	N	-
FOXTAIL, YELLOW	C	N	N	C	C	C		C	N	C
GOOSEGRASS	C	N	N	C	-	C		C	N	C
JOHNSONGRASS(Pr)	N	N	N	-	C	N		-	N	N
JOHNSONGRASS(Se)	C	N	N	P	C	C		-	N	C
OAT, WILD	C	N	N	P	C	C		-	N	P
PANICUM, FALL	C	N	N	C	-	C		-	N	C
RABBITFOOT, POLYPOGON	-	N	N	C	C	C		C	N	C
RESCUEGRASS	C	N	N	C	C	-		-	N	C
RYEGRASS, ITALIAN	C	N	N	C	C	P		-	N	C
SANDBUR	-	N	N	-	C	C		C	N	C
SPRANGLETOP, MEXICAN	C	N	N	-	C	N		-	N	C
STINKGRASS	C	N	N	P	-	P		-	N	C

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

Weed Susceptibility Chart

	TRIFLUSULFURON	2,4-D	2,4-DB
BROADLEAF			
BINDWEED(Pr)		P	N
BINDWEED(Se)		C	N
CARROT, WILD		-	-
CHICKWEED, COMMON		P	N
CLOVER		P	N
COCKLEBUR		C	C
CUDWEED		P	P
CURLY DOCK(Pr)		P	P
CURLY DOCK(Se)		C	C
DAISY, ENGLISH		-	-
DANDELION(Pr)		P	N
DANDELION(Se)		C	C
DODDER		N	N
FIDDLENECK		P	P
FILAREE		C	C
FLEABANE, HAIRY		C	C
FLUVELLIN		-	-
GOOSEFOOT, NETTLELEAF		C	C
GROUNDCHERRY		C	C
GROUNDSEL, COMMON		C	N
HENBIT		P	P
HORSETAIL		N	N
HORSEWEED, MARESTAIL		C	C
KNOTWEED		P	P
KOCHIA		C	C
KYLLINGA, GREEN		-	-
LAMBSQUARTERS		C	C
LONDON ROCKET		C	C
MALLOW, CHEESEWEED		P	P
MINERS LETTUCE		-	N
MORNINGGLORY, JAPANESE		C	P
MUSTARD		C	C
NETTLE, BURNING		P	P
NIGHTSHADE, BLACK		C	C
NIGHTSHADE, HAIRY		C	C
NUTSEDGE, PURPLE		N	N
NUTSEDGE, YELLOW		N	N
ONION, WILD		-	-
PEPPERWEED, PERENNIAL		P	N
PIGWEED		P	P
PINEAPPLE WEED		P	P
PLANTAIN, BUCKHORN(Pr)		P	P
PLANTAIN, BUCKHORN(Se)		C	C
PRICKLY LETTUCE		C	C
PUNCTUREVINE		C	P
PURSLANE		C	P
RADISH, WILD		C	C
REDMAIDS		-	-
RUSSIAN THISTLE		C	P
SHEPHERD'S-PURSE		C	C
SMARTWEED		P	-
SOWTHISTLE, ANNUAL		C	C
SPEEDWELL		-	-
SPURGE, SPOTTED		P	-
STARTHISTLE, YELLOW		C	P
SUNFLOWER, COMMON		C	C
SWINE CRESS		N	N
TANSY MUSTARD		C	C
THISTLE, CANADA		P	N
THORNAPPLE, CHINESE		C	P
TURKEY MULLEIN		P	-
VELVETLEAF		C	-
WILLOWHERB, PANICLE		C	-
WOODSORREL, CREEPING		P	-
GRASSES			
BARLEY, HARE		N	N
BARNYARDGRASS		N	N
BERMUDAGRASS(Pr)		N	N
BERMUDAGRASS(Se)		N	N
BLUEGRASS, ANNUAL		N	N
BLUEGRASS, BULBOUS		N	N
BROME, DOWNY		N	N
BROME, RIPGUT		N	N
CANARYGRASS		N	N
CEREALS, VOLUNTEER		N	N
CRABGRASS		N	N
DALLISGRASS(Pr)		N	N
DALLISGRASS(Se)		N	N
FESCUE		N	N
FOXTAIL, YELLOW		N	N
GOOSEGRASS		N	N
JOHNSONGRASS(Pr)		N	N
JOHNSONGRASS(Se)		N	N
OAT, WILD		N	N
PANICUM, FALL		N	N
RABBITFOOT, POLYPOGON		N	N
RESCUEGRASS		N	N
RYEGRASS, ITALIAN		N	N
SANDBUR		N	N
SPRANGLETOP, MEXICAN		N	N
STINKGRASS		N	N

Se = SEEDLING, "Pr" = PERENNIAL," -" = NO RATINGS," N" = NO CONTROL, "P" = PARTIAL CONTROL, "C" = CONTROL

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